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The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

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New York City, New York

JOSEPH L. BERNIER, D.D.S., M.S., F.D.S., R.C.S.
President, American Academy of Periodontology, 1959

Dr. Joseph L. Bernier, Colonel of the Dental Corps of the United States Army, was installed as President of the American Academy of Periodontology in Dallas, Texas, in November 1958.

Dr. Bernier is Chief of the Dental and Oral Pathology Branch of the Armed Forces Institute of Pathology, Washington, D. C., Professor of Oral Pathology at Georgetown University School of Dentistry, Pathologist to the Registry of Oral Pathology of the American Dental Association, Educational Advisor to the Army Dental School Walter Reed Army Institute of Research, Washington, D. C., and Lecturer in Oral Pathology to the Jefferson Medical College, Philadelphia, Pennsylvania. He has been very active in academic circles, has published six books, innumerable articles and appeared on most of the major dental society programs.

Dr. Bernier is past President of the American Association of Oral Pathology, a Diplomate of the American Board of Periodontology and the American Board of Oral Medicine, a Fellow in Dental Surgery of the Royal College of Surgeons in England, past President of the American Board of Oral Pathology, and a member of most of the scientific societies in this country and many abroad. He has received many honors, including the Certificate of Achievement from the Department of the Army, Louis Livingston Seaman Award of the Association of Military Surgeons, the Award for Leadership in Oral Pathology from Tufts University School of Dental Medicine, the Thomas P. Hinman Award, the John R. Callahan Award, the Alfred C. Fones Award, the Award of Merit from Georgetown University School of Dentistry, and many others.

In addition to Dr. Bernier's many other activities, he has had a great interest in the activities of the American Academy of Periodontology, finding time to serve on many of its important committees. He brings to the Presidency of the Academy many years of experience which should result in an increase in the influence of the Academy.

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY NINE

Alveolar Bone Loss as Related to Oral Hygiene and Age*

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Oslo, Norway

LOSS of alveolar bone is one of the cardinal symptoms of periodontal disease, and resorption of the alveolar crest can be evaluated roentgenologically. In an examination of an Indian population Marshall-Day and Shourie[1] observed alveolar bone loss in all persons above 17 years of age. The bone loss was most advanced around the front teeth and molars in the lower jaw, and least advanced around the premolars. The resorption of the alveolar crest was measured in tenths of the optimum bone height.

Belting, Massler and Schour[3] examined 5014 adult men in Chicago, and found that the resorption of the alveolar bone started before the age of 20-24 years. Bone loss was considered to have taken place when the alveolar crest was found 2 mm or more from the cemento-enamel junction.

Herulf[2] studied the relationship between alveolar bone loss, gingival changes, presence of calculus, and tobacco consumption in an examination of about 600 dental students. For the sake of accuracy the measurements were limited to the lower front teeth. Herulf found that the bone loss was significantly greater among the smokers than among those who did not smoke. Furthermore, the interdental bone septa were on an average lower in the groups with gingival changes and calculus formation than in the control groups. However, within the main group with gingival changes he did not observe any difference between the calculus-free and the calculus-susceptible persons.

To the best of our knowledge there has not been carried out any systematic investigation on the relationship between the efficiency of oral hygiene and the loss of alveolar bone. The present study represents an attempt to this end.

MATERIAL AND METHOD

The examination was carried out on a group of 737 male workers and staff members of a modern industrial plant in Oslo. Only age groups up to 45 years

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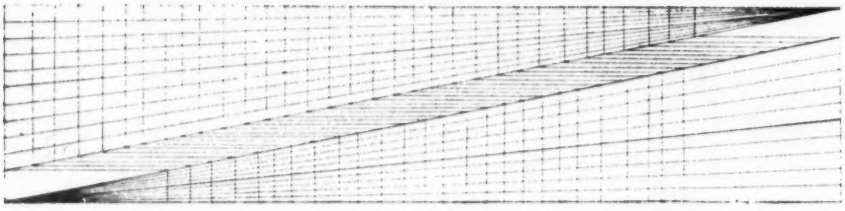


Fig. 1. Ruler for measuring percentage of bone loss in roentgenographs. See text.

were included in the examination. In the older age groups the loss of teeth was too large to allow systematic measurements to be made. Besides that, many of the missing teeth were obviously extracted on account of periodontal disease; for that reason the assessment of the average bone height around the remaining teeth would not give a representative picture of the condition. The material was divided into the following age groups: 21-25, 26-30, 31-35, 36-40 and 41-45.

A set of 10 roentgenographs was taken from each person, and in these the bone height was measured in percentage of "maximum bone height." On the basis of Herulf's investigation, and on the basis of observations in histologic sections from normal human beings, the alveolar crest was considered to be of optimum height when it was found 1 mm or less from the cemento-enamel junction. Consequently, bone loss was considered to be present only in those cases where the alveolar crest was found more than 1 mm from the cemento-enamel junction.

For several reasons the system of Marshall-Day and Shourie, who measured the bone loss in percentage of maximum bone height, was adopted. These authors, however, have not described the equipment they used for the measurement; therefore, a special measuring gadget was devised (Fig. 1). On a translucent plastic ruler a line was made 1 mm from the margin corresponding to the "normal" distance from the alveolar crest to the cemento-enamel junction. Close to the end of this baseline a point was chosen as center, and from this center 10 radii with exactly the same distance between each of them were made; the baseline as well formed a radius—the first one.

During the measurement the ruler was placed over the roentgenograph in such a way that the margin of the ruler covered the cemento-enamel junction. The ruler was then moved until the last radius covered the apex (Fig. 2). Through the translucent ruler the alveolar crest could be seen under one of the radii or between two

Fig. 2. Ruler described in Fig. 1, placed on top of roentgenograph in order to measure percentage of bone loss on distal surface of lower right central incisor. Bone loss 20%. See text.

of them. In that way it was possible to assess the height of bone with an accuracy of 1/20. As alveolar crest was considered the point where the periodontal membrane space was found to be of normal, or approximately normal, width. However, in many cases such a definite point could not be found, for which reason no measurement was made. Where no interproximal fillings were present, the cemento-enamel junction could be localized with a reasonable degree of certainty in most cases, although difficulties were encountered in others. In many cases the cemento-enamel junction was obscured by interproximal fillings, and if so, the cervical margin of these fillings were chosen as the baseline. However, when the fillings obviously were located below the cemento-enamel junction the bone height was characterized as "unmeasurable."

The measurements were carried out in good translucent light, and the reader used a powerful lens. Separate measurements were made for the mesial and distal surfaces of each tooth.

In about 25% of the bone septa no measurement could be carried out. This was mostly due to overlapping of the teeth and difficulties in assessing the alveolar crest or cemento-enamel junction. In the upper jaw the roentgenographs were taken with an angulation of 35, 45 and 55 degrees for the molars, premolars and the front teeth respectively, and the central rays were directed against the collum of the teeth. To test the influence of the angulation for the results of the measurements, a number of roentgenographs were taken with an angulation of 10° more and 10° less than the standard type. In the anterior region this gave little difference in the results. However, in the molar region differences of up to 20% could be observed. Roentgenographs with too acute an angulation were, consequently, disqualified. Overlapping was most commonly found in the cuspid or lateral incisor area.

At the same sitting as roentgenographs were taken, a clinical examination of the patient was undertaken, and among the clinical data were charted the efficiency of oral hygiene. The toothbrushing was considered to be (1) good, when all, or practically all, surfaces accessible to the toothbrush were clean, and (2) fairly good, when some of the surfaces accessible to the toothbrush were clean, and (3) not good, when practically all the surfaces accessible to the toothbrush were covered with deposits that could have been removed if the brushing had been effective.

The distribution of the material according to these criteria is seen in Table I.

TABLE I

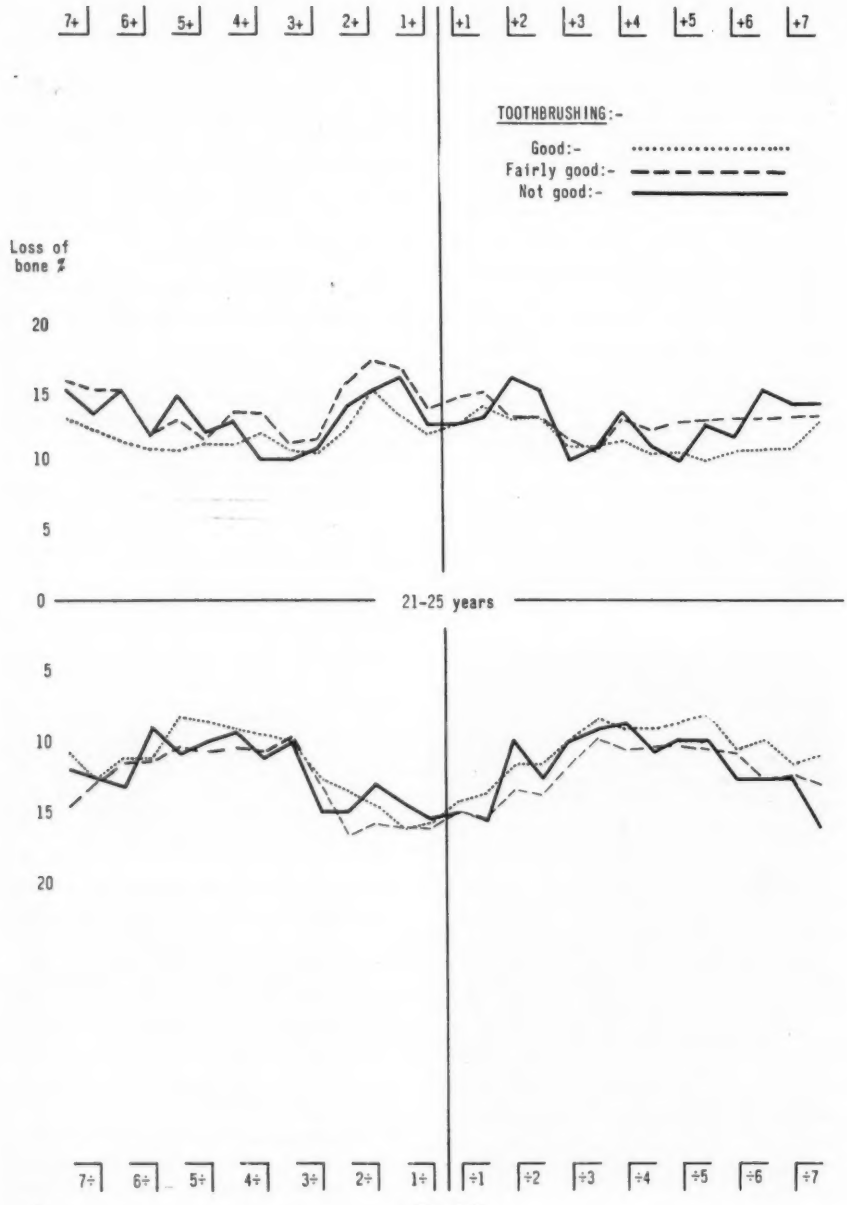
	Efficiency of Tooth-Brushing			
Age in years	*Good*	*Fairly good*	*Not good*	*Total*
21 - 25	13	59	9	81
26 - 30	24	101	23	148
31 - 35	24	139	54	217
36 - 40	14	89	64	167
41 - 45	10	58	56	124
Total	85	446	206	737

The interpretation of the roentgenographs was carried out as a blind test, i.e., the one studying them did not know to which group of hygiene the person belonged, neither had he taken part in the clinical examination of the patients.

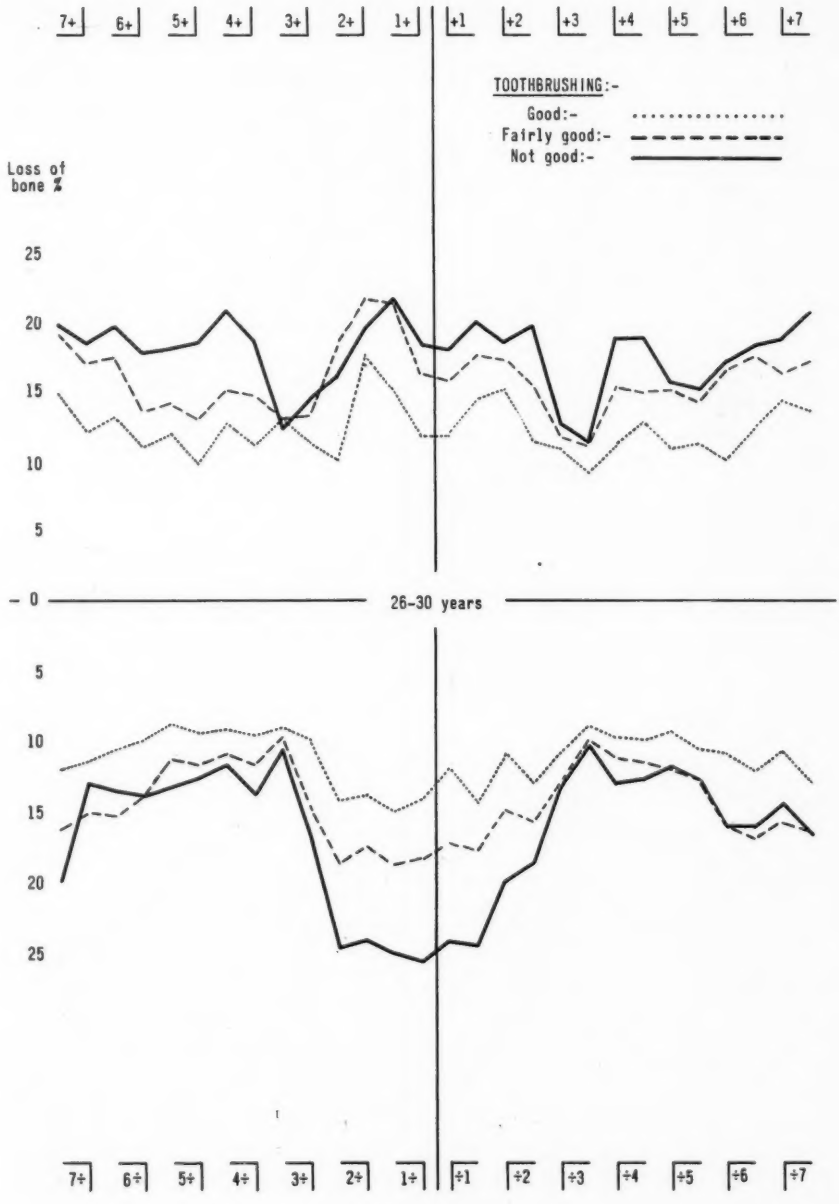
In each age and hygiene group the figures for the bone height of the individual tooth surfaces were summed together and divided with the number of measured surfaces. This gave the average bone height around the remaining teeth. These values were plotted on a diagram for each jaw.

FINDINGS

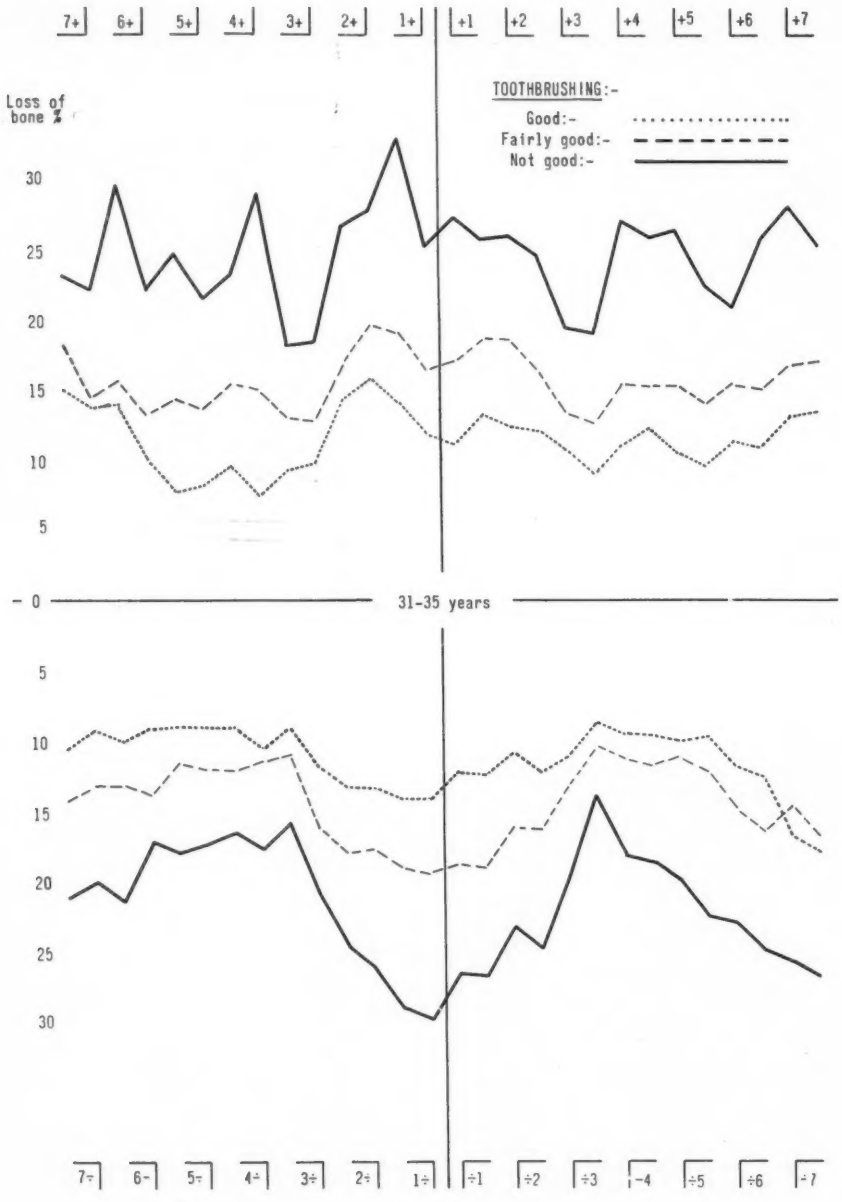
In the age group 21-25 years, the difference in average bone height between the three hygiene groups is not considerable (Graph I), but the group with good hygiene tends to have lost less alveolar bone than the two other groups.


7+
6+
5+
4+
3+
2+
1+
+1
+2
+3
+4
+5
+6
+7
TOOTHBRUSHING:-
Good:-
Fairly good:-
Not good:-
Loss of bone %
20
15
10
5
0
21-25 years
5
10
15
20
7÷
6÷
5÷
4÷
3÷
2÷
1÷
÷1
÷2
÷3
÷4
÷5
÷6
÷7

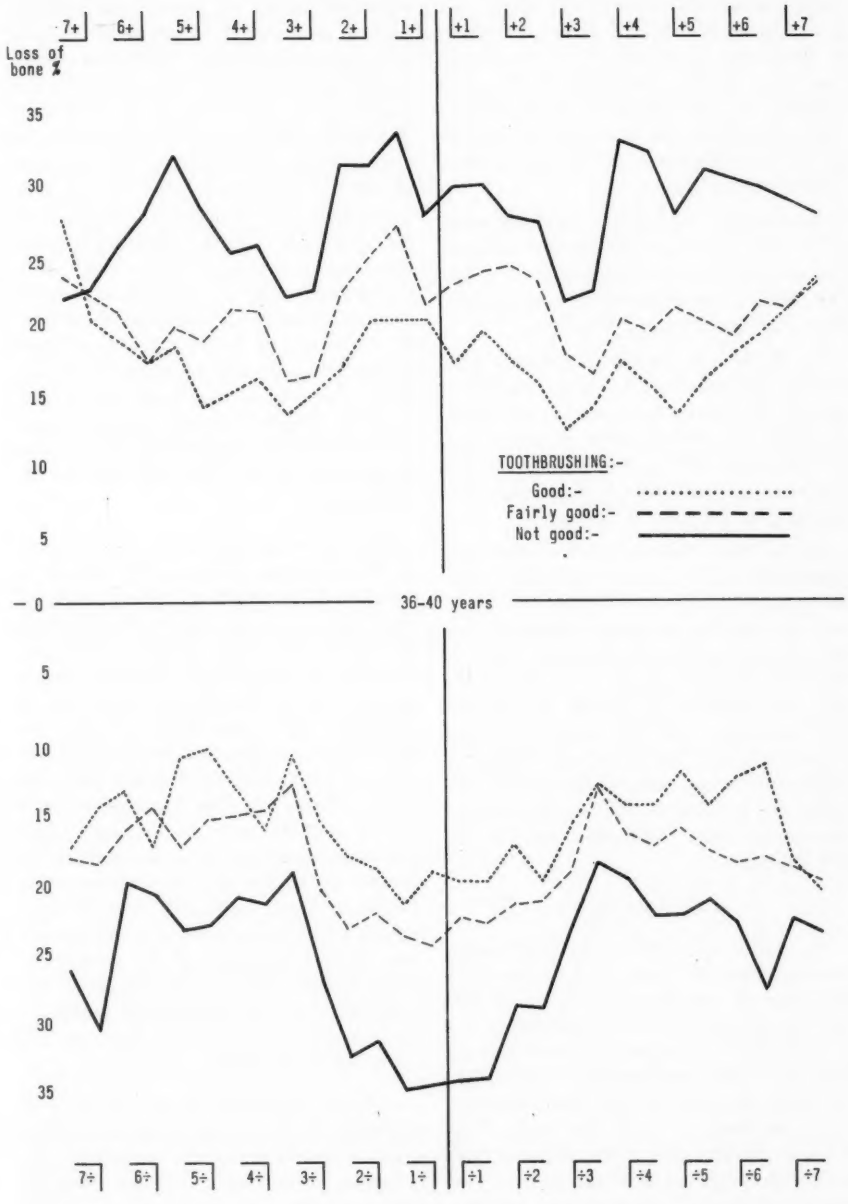
Graph I


7+ 6+ 5+ 4+ 3+ 2+ 1+ +1 +2 +3 +4 +5 +6 +7
TOOTHBRUSHING:-
Good:-
Fairly good:-
Not good:-
Loss of bone %
25
20
15
10
5
0
26-30 years
5
10
15
20
25
7÷ 6÷ 5÷ 4÷ 3÷ 2÷ 1÷ ÷1 ÷2 ÷3 ÷4 ÷5 ÷6 ÷7

Graph II


7+
6+
5+
4+
3+
2+
1+
÷1
÷2
÷3
÷4
÷5
÷6
÷7
Loss of bone %
TOOTHBRUSHING:-
Good:-
Fairly good:-
Not good:-
30
25
20
15
10
5
0
31-35 years
5
10
15
20
25
30
7÷
6÷
5÷
4÷
3÷
2÷
1÷
÷1
÷2
÷3
÷4
÷5
÷6
÷7

Graph III


7+
6+
5+
4+
3+
2+
1+
+1
+2
+3
+4
+5
+6
+7
Loss of bone %
35
30
25
20
15
10
5
0
TOOTHBRUSHING:-
Good:-
Fairly good:-
Not good:-
36-40 years
5
10
15
20
25
30
35
7÷
6÷
5÷
4÷
3÷
2÷
1÷
÷1
÷2
÷3
÷4
÷5
÷6
÷7

Graph IV

The difference between the three hygiene groups comes out more clearly in the age group 26-30 (Graph II), and even more so in the groups 31-35 and 36-40 (Graphs III and IV). In the age group 41-45 years (Graph V), the difference is conspicuous and systematic in the lower jaw; in the upper jaw the differences are obvious, but not always systematic.

When the 5 age groups are compared with each other, continuous loss of bone can be observed in all hygiene groups, but the speed is conspicuously higher among those having a poor oral hygiene.

In all the age groups the average percentage of bone loss tends to be higher around the anterior teeth, both in the upper and lower jaw. Next in order are the molars and premolars. The highest bone percentage was usually found around the cuspids, particularly on their distal surfaces.

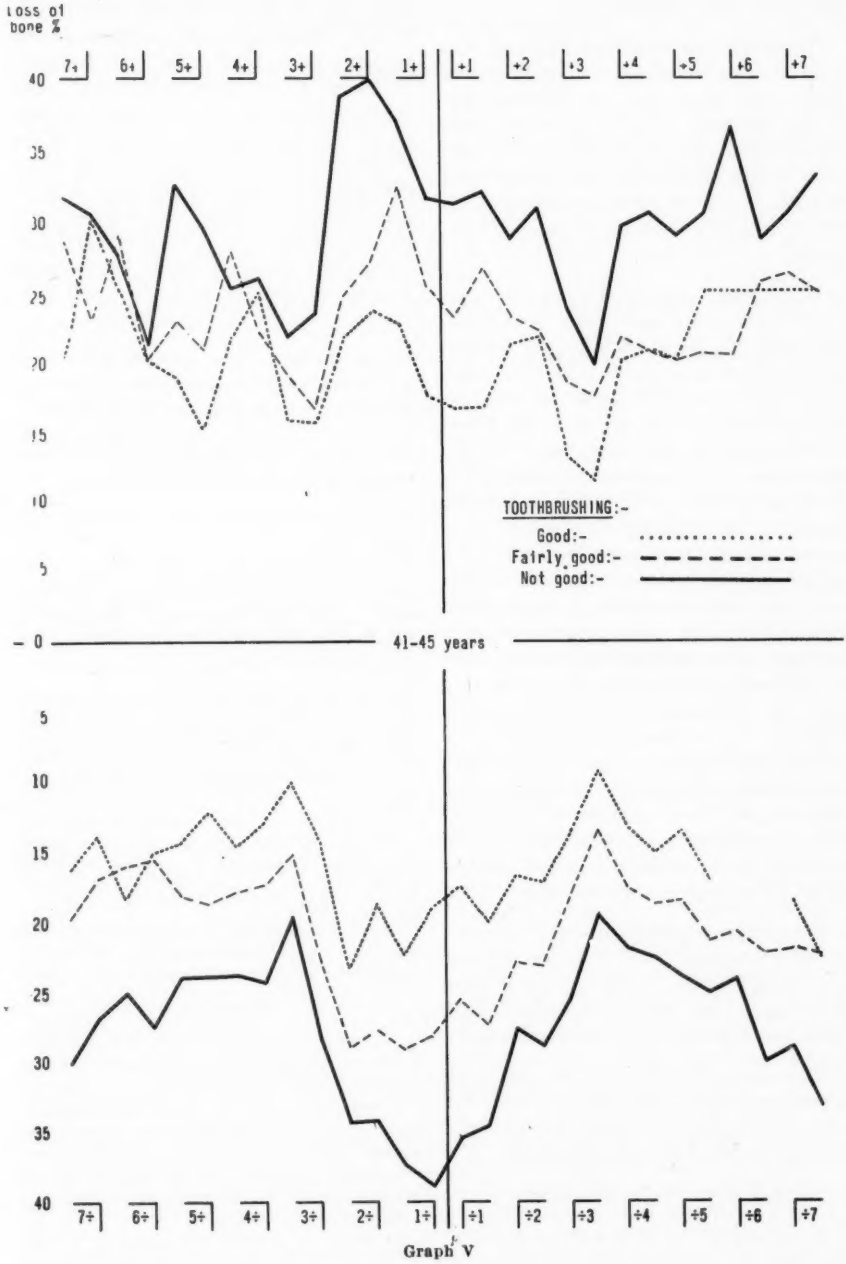
In the molar, bicuspid and cuspid area, the bone level was rather symmetrical, with no systematic difference between the right and left side. In the anterior however, the bone percentage was significantly lower on the right side than on the left. This tendency was observed in all the age groups and manifested itself in nearly all of the hygiene groups. The asymmetric bone resorption in the anterior was seen both in the upper and lower jaws, and usually the difference was most conspicuous on the lateral incisors.

DISCUSSION

This statistical investigation clearly demonstrates that the destruction of the alveolar bone is significantly related to the efficiency of oral hygiene. Besides being related to the oral hygiene, the bone resorption increased continuously with age. However, the speed of the bone resorption cannot be deducted from this study. This is due to the fact that those teeth that had been most subjected to destruction of the supporting bone, had been extracted because of this destruction. Therefore, at the time of the examination the bone level around these teeth might have been zero or at least very low. As the number of teeth extracted because of periodontal involvement is unknown, it is impossible to state how much these teeth would have influenced the mean values. It is not likely that extraction of teeth because of periodontal involvement has influenced the mean values in the youngest age group to any mentionable degree. Such an influence is, however, quite obvious in the age group 41-45 years and explains the slight difference between this group and the age group 35-40 years. Extraction because of periodontal involvement may also explain some of the irregularities in Graph 5. On the basis of these considerations it can be stated with certainty that the average bone levels illustrated in Graphs 1-5, give too favourable an impression of the periodontal conditions —the more so the older the persons examined are.

Also for other reasons the figures given in this examination cannot be claimed to represent absolute values, and a number of reservations must be taken: (1) The normal distance between the cemento-enamel junction and the alveolar crest was chosen more or less arbitrarily and may not be altogether right. (2) The location of the cemento-enamel junction is often difficult to decide with certainty; this may partly be due to fillings. (3) The bone resorption is usually most advanced on the lingual and approximal surfaces, and it was not always possible to decide the exact bone level. (4) In the upper jaw in particular, the film cannot be placed parallel with the tooth; this implies distortion of the roentgenograph, and the effect of this may go in one direction. This may explain the comparatively low bone level in the front in the age group 21-26 years.

Although the figures cannot be claimed to give an altogether correct picture of the situation within any of the age groups, the technique is sufficiently reliable when it comes to comparing the bone level in the three different hygiene groups. In this case the influence of the method error will be the same in all the three categories, and as


Loss of bone %
7+
6+
5+
4+
3+
2+
1+
+1
+2
+3
+4
+5
+6
+7
40
35
30
25
20
15
10
5
0
TOOTHBRUSHING:-
Good:-
Fairly good:-
Not good:-
41-45 years
5
10
15
20
25
30
35
40
7÷
6÷
5÷
4÷
3÷
2÷
1÷
÷1
÷2
÷3
÷4
÷5
÷6
÷7

Graph V

blind test was employed, any subjective judgment was excluded.

Therefore, the beneficial effect of oral hygiene is convincingly established in this study. It is likely that the difference between the three hygiene groups is even greater than it appears from the graphs. This is due to the fact that many more teeth had been extracted because of periodontal disease in cases with poor oral hygiene.

The conspicuous asymmetry of bone level in the front needs some further explanation. To begin with it was difficult to understand why such a difference manifested itself. It could hardly be explained as a method error, as the asymmetry was systematic in all the age groups. The explanation of this apparent discrepancy has probably been given by Hirschfeld. In his study of the toothbrushing habits of a great number of patients Hirschfeld observed that they skipped the right front teeth when they turned the brush from the right to the left side, or vice versa. By checking the toothbrushing among some of the persons belonging to the present material, the authors observed that such a tendency was predominating among right-handed persons; and as right-handed persons in any great group will represent the majority, it is to be expected that the uneven efficiency of the toothbrushing should manifest itself statistically in a difference in the bone level on the two sides.

It may appear surprising that this asymmetry manifested itself even among the poor toothbrushers. However, the persons belonging to this group who brushed their teeth at all, brushed the front teeth only.

The observation that the alveolar bone resorption is most advanced in the front and molar region is in agreement with the findings of Marshall-Day and Shourie.

SUMMARY AND CONCLUSIONS

In a group of 737 male workers and staff members of a modern industrial plant in Norway, the influence of oral hygiene on the resorption of alveolar bone was evaluated. The persons were classified in three groups according to the efficiency of oral hygiene: good, fairly good and not good. A set of 10 roentgenographs was taken from each person, and in these the percentage of bone loss was measured on the mesial and distal surfaces of all the teeth, providing that the pictures allowed measurements to be made. Optimum bone height was considered to be present when the alveolar crest reached 1 mm or less from the cemento-enamel junction. A special ruler was designed for the measurements.

The means of the measurements were calculated and illustrated in Graphs 1-5. These conclusions seem to be justified.

(1) Loss of alveolar bone increases with decreasing efficiency of oral hygiene.

(2) Loss of alveolar bone increases with age.

(3) The alveolar bone around the teeth seems to be subjected to resorption in the following order: anterior teeth, molars, bicuspids and cuspids.

(4) Right-handed people usually seem to brush the left incisors better than the right ones. This asymmetric toothbrushing manifests itself in more bone loss on the right side.

Acknowledgements. The authors like to express their sincere thanks to Dr. Nygaard Ostby, Head of the Norwegian Institute of Dental Research, for excellent cooperation.

The statistical evaluation has been carried out by Mr. Sven Vigger of the University Institute of Economics, University of Oslo.

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Bone Graft in the Treatment of Intrabony Periodontal Pocket in Dogs
A Histological Investigation*

by Isra Yuktanandana, d.d.s., m.s., *Birmingham, Alabama*

BONE graft has been successfully utilized in the medical field for a number of years, and the use of grafts to fill osseous defects of the jaw bones and to raise the height of the edentulous mandible have been successfully reported by many investigators. However, there is only a small amount of literature on its use in periodontology.[1-13] In experimental works,[1-5] it appears that bone regeneration and reattachment can be induced in surgically produced periodontal pockets in animals by the insertion of bone, cartilage, cementum, or dentin implants. But since the work of Hegedus,[7] and Beube and Silvers,[6] there is no clinical report regarding its use in periodontal patient until recently. However, Beube[8] suggests the use of boiled bone powder in the flap operation for intrabony pockets of 7mm. depth or greater, around single-rooted teeth which have not been closed by curettage. In the last three years, more literature on the subject has appeared.[9-13] Cross,[9,10] Forsberg[11] and Krømer[12] have reported successful results in patients using bone transplantation for the treatment of intrabony pockets. According to these reports, the pocket depth was markedly reduced and in many instances a normal gingival crevice was formed, but whether there was a new attachment of periodontal fibers between the new bone and the root surface, or whether there was a close adaptation of the epithelial attachment along the tooth surface has not been determined.

It is the purpose of the present investigation to study the histological appearances of the grafts in intrabony pockets for varying periods of time and to determine whether healing takes place by reattachment or readaptation. At the same time the findings may throw light on some aspects of bone transplantation under this particular circumstance and suggest an improvement of operative technique.

MATERIALS AND METHOD

In order to investigate the healing of bone graft in an intrabony pocket, the present experiment had to be performed in two parts. The first part was the creation of an intrabony pocket, and the second was the bone graft operation.

A total of ten dogs of both sexes was used. These animals were of unknown age and parentage, and were picked at random. Some of the animals were healthy, some had gastro-intestinal disturbances, and one contracted distemper and had to be discarded. The periodontal condition of these animals varied. Only two dogs had clinically healthy gingiva. The others had some form of periodontal disease which ranged from mild marginal gingivitis to severe periodontitis with pocket formation.

The Creation of Intrabony Pockets. The creation of periodontal pockets in animals has been reported by many investigators.[1-5,14-16] Schaffer[3,4] has reported a technic of producing surgical intrabony pockets, but at the present time no report concerning the creation of pathological intrabony pockets has been published. In the present experiment, the intrabony pocket was created surgically. In order to simulate pathological pockets in man, it was treated by various materials and was left for one week until pathological changes occurred. The pocket produced by this method differed from the pathological intrabony pocket in man in that it was more acutely inflamed, and the epithelial lining of the pocket wall had not yet been established.

The pockets were produced on both sides of the maxillary arch of these animals; one for the experimental graft, and the other for control. Because of the shape and arrangement of dog's teeth, it was found that the interproximal areas between the

*Part of a thesis submitted in partial fulfillment of the requirements for the degree Master of Sciences in Dentistry at the University of Alabama. This project was directed by Dr. Gilbert J. Parfitt, Head of the Department of Periodontology, School of Dentistry. Abstract was presented at the meeting of the International Association for Dental Research, Detroit, Mich., March 1958.

canine and the first premolar, or between the first and second premolars were the most suitable. After the animal was anesthetized by intravenous injection of veterinary Nembutal Sodium, the clinical condition of the gingiva was observed. If there were any signs of periodontal disease, or if calculus was present, scaling and curretage were performed in the operating areas prior to the operation for pocket formation.

A vertical incision was made mesial to the area, starting from the gingival margin down to the mucobuccal fold. After the mesio-distal interdental incision had been made, buccal and palatal flaps were laid back to expose the interdental septum. A 701 or 702 fissure bur was then used to create a V-shaped intrabony pocket along the mesial surface of the tooth. The use of engine-driven instruments in this experiment was always accompanied by water spraying to reduce heat. The depth of the bony part of the pocket varied from 3 to 4 mm., depending upon the length of the root. The mesio-distal width of the opening was about 2 mm. The buccal and palatal alveolar plates on each side of the pocket were also removed to approximately one fourth of the depth of the pocket. The exposed root surface was then curetted and filed to remove the remaining soft tissues. A notch in the root was then made with a small bur at the bottom of the pocket as a point of reference. As bone regeneration often occurs following a surgically produced pocket, the space so created was filled with self-curing acrylic, tin foil, or surgical cement, before the flaps were coaptated and sutured.

The clinical condition of the pockets, after a period of one week, differed a great deal depending on the type of the materials inserted. Self-curing acrylic for the making of models was found to be unsuitable as it produced a large area of necrosis of both the bone and soft tissue. The animals treated with this material were excluded from the experimental series, except one, in which the buccal and palatal alveolar plates were retained.

Since the use of self-curing acrylic produced such a large amount of inflammation and bone loss, tin foil was tried. A piece of tin foil was molded to conform to the shape and size of the pocket before insertion into the surgically created pocket. One week after the operation, the pocket areas showed little inflammation. After the tin foil was taken out, it was found that the pocket areas were a little wider and deeper than was made surgically, but not as much as that resulting from packing with acrylic. In the cases in which only thin buccal plates were left after surgery, these parts were usually retained at the bone graft operation, but sequestration occurred approximately one month later.

A surgical cement called "Wonderpak" was tried in the creation of the intrabony pocket involving the bifurcation. The results seemed to be better than when tin foil was used. Although the pockets appeared a little wider and deeper after one week, there was very little gingival inflammation. However, all of the intrabony pockets in this experimental series were treated with tin foil except in one dog.

In every case two pockets were created in the same jaw of each animal, one on the right side for bone graft experiment, and one on the left for control. The operations were similar on both sides, but the size and shape of the pockets in different dogs varied, depending upon the length of the root, the thickness of the interseptal bone, and severity of post-operative inflammation.

The Bone Graft Operation. The bone graft operation was performed one week after the pocket was surgically created. The animals were anesthetized by intravenous Nembutal Sodium. In addition, local infiltration of two per cent xylocaine hydrochloride with epinephrine 1:100,000 at the operating sites was also given.

The Donor Area. Fresh autogenous cancellous bone was used in the present series of experiments. After studying sections made of the dog's skull, it was found that a suitable area to provide bone for such a graft was between the maxillary precanine and the canine. In this area the cortical plate is not too thick, and there is enough cancellous bone beneath it to be used for the operation. In addition, this particular area gives better access, and the cancellous bone can easily be removed through a window in the cortical plate on the buccal surface.

After retraction of the mucoperiosteum, a trephine of 5 mm. diameter was used to cut a window in the cortical plate. When the cortical plate was removed, the same trephine was used to obtain the deeper cancelous bone. This bone was removed and place in saline solution, then cut into several small pieces by scissors for transplantation into the prepared pocket. The flap was then replaced and sutured.

The Pocket Areas. After the muco-periosteal flaps were raised and the packing material removed, the whole pocket, including the root surface, was carefully curetted and filed. This was to remove necrotic tissues in the pocket and to freshen the bone surfaces. Blood clot was then allowed to form in the pocket. As the graft bed was prepared, the cancellous bone chips were inserted into the pocket, piece by piece, building from the bottom of the pocket until the level of the original alveolar crest was almost reached. Care was taken to see that all the chips were covered with blood clot before the flaps were replaced and sutured.

The operation on the control side was done at the same time as the grafting operation. The packing material was removed without raising the flaps, and thorough curettement of the pocket was done by curettes and files. In fact, the procedure was the same as on the experimental side, except for the insertion of the bone chips. Blood clot was then allowed to form. Since the flaps were not raised on the control side, no suturing was necessary.

Post-Operative Care. The dogs were fed on skim milk for one week after the grafting operation, they were then returned to a normal diet. The suture was removed one week after the operation.

To observe the healing process of the bone graft in the intrabony pocket, the animals were sacrificed at one, two, three, eight, twelve and sixteen weeks after grafting. The tissue block including the teeth mesial and distal to the intrabony pocket was immediately removed and prepared for microscopic examination. Sections were stained with hematoxylin-eosin.

EXPERIMENTAL RESULTS

All animals survived the experimental period but three had to be discarded. One of the dogs became severely ill, in another the wounds remained open and all the grafts were exfoliated leaving the exposed bony wall of the pocket visible; and in the third, accidental pulp exposure occurred during the creation of the pocket. Although in this last case the healing of the wound was clinically normal and a large portion of the graft was successful, the process of healing was considered to be somewhat different from the other animals.

The clinical condition of the pocket and the surrounding gingivae were observed throughout the entire experimental period. Roentgenograms of the pockets were taken before and after the insertion of the graft and at the end of the experiment.

For simplicity, the findings in the one, two and three weeks specimens will be reported together, and similarly, the findings at the end of eight and sixteen weeks.

Clinical Observations. In an early experimental period the rate of the healing of the gingiva on the experimental side was slower than that usually seen in humans. In some animals, the sutures were lost after the first few days, and the margins of the wounds gaped open. Some of the grafted bone usually presented into the mouth. However, the remaining portions of the grafts were incorporated within the granulation tissue. The surrounding gingiva was slightly inflammed. In two weeks, the line of incision was still present and showed signs of inflammation. The inflammation became much less in three weeks except in the area surrounding a protruding graft. The healing of the gingiva on the control side presented almost the same clinical condition as on the experimental side. But there was less inflammation and the rate of healing was somewhat faster than the experimental side. Various degrees of gingival recession occurred on both sides, but there was more on the control than on the experimental side.

In some dogs it was noticed that there were inflammatory areas on the buccal surface on both sides. This inflammation lasted for more than a month after the graft operation. On some occasions a piece of bone, larger than the size of the bone chips, protruded through the buccal gingiva. It is possible that this was sequestration of the thin buccal alveolar wall of the pocket following the irritation at the time of pocket formation. On the contrary, no inflammation or sequestration has been observed on the palatal side. This may be due to the fact that the palatal alveolar plate is thicker.

The healing of the gingiva in the later periods of the experiment was the same. All the wounds in both the experimental and control sides were healed by eight weeks. They presented the same appearance as in any interdental area in the mouth, except for some gingival recession.

No measurement of the pocket depth was carried out in the first three weeks as it was felt that insertion of the probe into the pocket would not only interfere with healing but might distort the tissues. However, the depth of the crevice after the areas had healed was measured. The depths of the crevices were approximately 1 mm. on both the experimental and control sides.

Roentgenographic Observations. Roentgenograms of the pockets were taken before and after the insertion of the graft and at the end of the experiment.

In the early periods of the experiment, not much change was seen in the roentgenograms. In the animal sacrificed at the end of two weeks, the radiolucent area of the pocket on the control side before operation and two weeks after, appeared to be almost the same (Fig. 1 a and b). The roentgenograms taken immediately after the operation showed the presence of radio-opaque area of the graft (Fig. 1c), and the roentgenograms two weeks later showed almost the same picture (Fig. 1d). A small radio-opaque area of the uppermost piece of the graft being exfoliated can be seen.

In the later period of experiment marked differences can be seen between the roentgenograms taken before the operation and those taken at the end of the experimental period. In one sixteen weeks animal the higher level of the dense radio-opaque area due to formation of bone is apparent (Fig. 2b), in contrast to the one taken before the graft was inserted (Fig. 2a). In another sixteen weeks animal, the differences in height of the alveolar crests between the experimental side (Fig. 2c) and the control side (Fig. 2d) is also apparent.

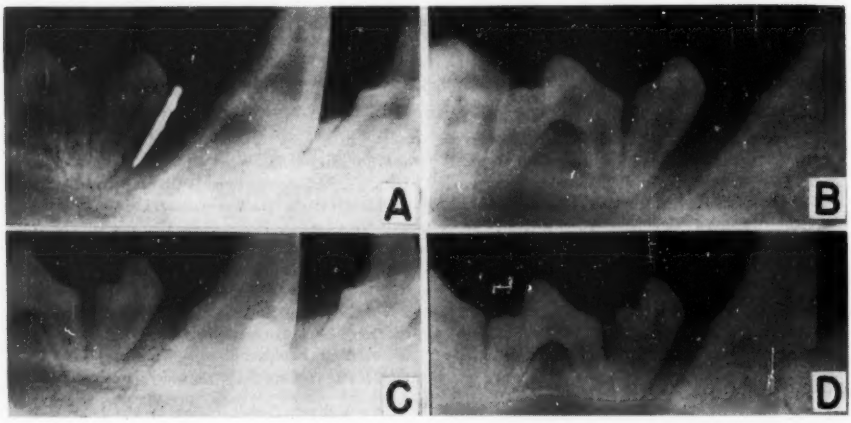

Fig. 1

HISTOLOGICAL OBSERVATIONS

1. Periodontal Regeneration: During the surgical procedure for pocket formation, a notch was made by a bur in the root of the tooth at the bottom of the pocket to serve as a point of reference for subsequent regeneration or recession of tissues. Coronal to the notch, the root surface was laid bare, alveolar bone and periodontal membrane was removed, and the root surface was curetted in order to free it of any adhering soft tissue. This curettage also removed all or most of the cementum from the root surface.

In histological sections, therefore, any bone observed coronally to the notch must have been formed since the notch was made. Similarly, any cementum laid down on the prepared root surface and containing periodontal fibers must also have been laid down since the time of operation.

Measurement of the exact amount of bone formation is difficult, for sections may not be exactly vertical or parallel to the long axis of the tooth. However, tissue blocks were carefully oriented before sectioning, and only those sections which closely approximated the vertical were used in this part of the study.

Figure 3 is a photomicrograph from the experimental side in an eight-week experiment. It shows a notch in the root at the base of the created pocket with new bone, new periodontal fibers, and new cementum being formed. The notch is almost completely filled with new cementum. In this section approximately 2.5 mm. of reattachment and 2.0 mm. of new bone have been formed. The bone is

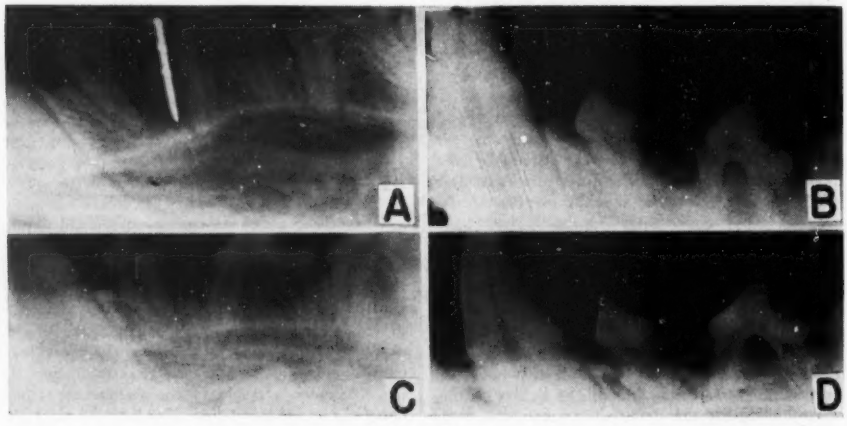

Fig. 2

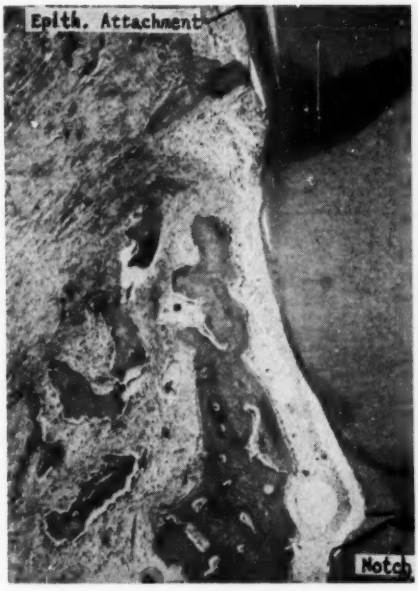

Fig. 3

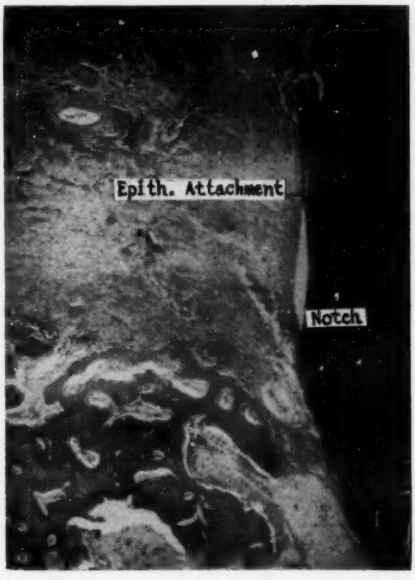

Fig. 4

still actively growing, and new bone formation on the surfaces of the grafts is also apparent.

The photomicrograph of the control side is shown in Figure 4. There is a long downgrowth of the epithelial attachment close to the notch area. The alveolar crest is at the level of the lower border of the notch. The greatest amount of periodontal regeneration seen in this control series is about 1 mm. in depth.

The result in the sixteen-week experiment is shown in Figure 5. There is approximately 3 mm. of new cementum deposition and connective tissue reattachment. The height of the alveolar bone is 2.5 mm. above the lower level of the notch. In an area not shown in the picture, a piece of graft is seen covered with new bone and joined to the new bone formation without any evidence of remodelling changes.

On the contrary, the control side of the sixteen-week experiment shows less than 1 mm. of periodontal regeneration (Fig. 6). But in general, the healing is proceeding normally. The epithelium is healed and keratinized, and is free from any cellular infiltration except at the area adjacent to the gingival crevice. Serial sections show that epithelium proliferates down to the level of the alveolar crest and less than 1 mm. above the lowest margin of the notch.

An almost complete reattachment with new bone formation is shown in Figure 7. This is a specimen from an eight-week dog, and the pocket was produced in the usual manner under the bifurcation of the premolar tooth. This was intended to resemble a grade II bifurcation involvement, according to Glickman's classification.[17] The opening on the buccal side was about 2 mm. wide, but this small opening led to an area at least 3 mm. across the bifurcation. The soft tissue adhering to the surface of the root was removed by curettes and the space was packed with surgical cement. After a week, the surgical cement was removed, and the bone graft was inserted in the usual manner. Unfortunately, this tissue block was cut obliquely, and the amount of periodontal regeneration can not be measured. But it is evident that the pocket is filled with connective tissue, new periodontal fibers are attached to the cementum, and osteoid tissue is forming on the graft. The epithelial attachment is in close adaptation to the bifurcation of the tooth except in the area of the opening on the buccal surface.

II. Incorporation of the Bone Graft into the Healing Wound: The graft site one week after the operation shows inflammed tissue in the upper half of the pocket. All of the grafts are undergoing osteoclastic resorption. Some are exfoliating, and are surrounded by the epithelium. Some parts of the epithelium are necrotic and infiltrated with inflammatory cells, in which polymorphonuclear leukocytes are the most predominant. The epithelial attachment proliferates apically to the lowest level of the inflammatory tissue, which is about half the depth of the pocket. The lower half of the pocket is filled with uninflamed granulation tissue of repair. The parent bone at the base of the pocket shows

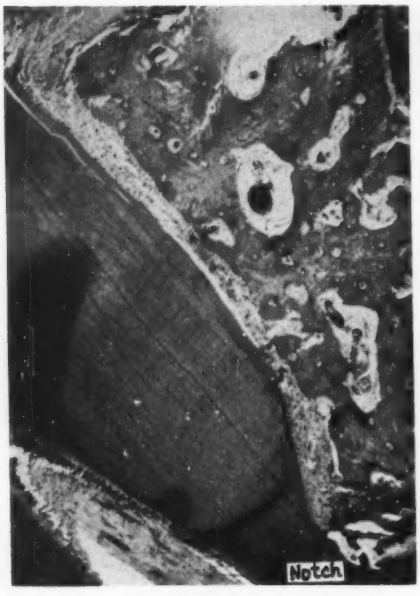

Fig. 5

Fig. 6

Fig. 7

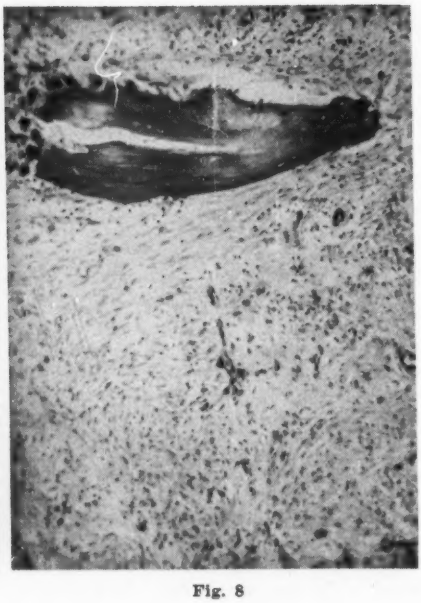

Fig. 8

active new bone formation with evidence of new spicules and radiating fibers passing into the surrounding connective tissue. Figure 8 shows the appearance of the graft which lies close to the parent bone. There is more osteoclastic activity on the upper surface while the undersurface is in close approximation with young connective tissue cells. Osteoid formation is not yet evident. Most of the lacunae in the grafts are empty, only a few osteocytes are present in some of the grafts.

The lowest level of the notch in the root of the tooth is usually below the level of the rapidly growing parent bone. The notch itself is filled with connective tissue cells which lie in close approximation to the root surface.

In the two-week specimen, the overall picture of the upper half of the pocket is similar to that at the end of the first week. Polymorphonuclear leukocytes are predominant in the epithelium and lamina propria while lymphocytes and plasma cells are present in the deeper area. In the lower half of the pocket bone formation, both from the parent bone and from the grafts is more pronounced. Figure 9 shows new bone formation on the undersurface of a graft. Osteocytes are present in the lacunae of the new bone. A trabecula is forming from beneath the graft and appears to join with another which grows from the parent bone. Revascularization of the graft is evident. Radiating fibers around the grafts are also more pronounced but osteoclastic activity continues. Along the root surface the new deposition of cementum is not yet apparent.

The three-week specimen shows more inflammation than any other specimens. The upper three-fourths of the pocket area is filled with dense accumulation of inflammatory cells. Some of the grafted bone is being exfoliated, but a few portions show radiating fibers and active bone formation. Cementoblasts and a thin layer of new cementum are apparent on the root surface. However, the progress of healing appears to be at the same stage as seen in the two-week specimen. This is probably due to the greater amount of inflammation.

In the eight-week specimen, cellular infiltration appears only in the crevicular areas and around some of the grafts. More bone formation is evident in the lower half of the pocket. Osteoclastic activity continues, and individual pieces of graft can still be distinguished. On the root surface, thick cementum deposition is clearly seen both within and above the notch.

A similar picture appears in the intrabony pocket under the bifurcation after eight weeks. An island of necrotic graft is present, surrounded by inflammatory cells including lymphocytes, plasma cells and histiocytes. But close to this area, radiating fibers with new bone formation are still evident. A few scattered small pieces of graft show new bone formation on their surfaces. However, it is impossible to distinguish any grafts incorporated in the new

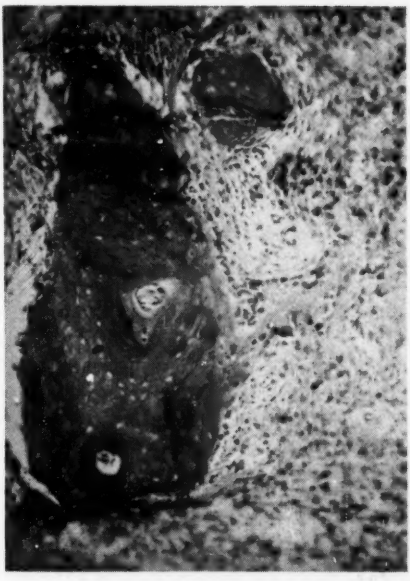

Fig. 9

bone formation. It is possible that the process of remodelling had been responsible for the removal of incorporated portions of the graft. Along the surface of the roots, a greater area of connective tissue reattachment is established. Only about 1 mm. width of epithelial attachment can be seen attached to the undersurface of the bifurcation (Fig. 7).

The sixteen-week specimen shows the appearance of normal tissues. The epithelial surface is keratinized. Cellular infiltration is present only in the lamina propria of the gingival crevice. There is a large area of connective tissue reattachment as shown in Figure 5. The transeptal fibers formed between the two teeth are oriented normally and are of mature size. Beneath the transeptal fiber there is normal alveolar bone. However, osteoblastic activity seems to be a little more than normal, and a few osteoclasts appear on the upper surface of the alveolar crest.

On the control side, in the absence of the graft, rapid bone formation from the parent bone occurs in the first few weeks. But at the same time, rapid apical migration of the epithelial attachment also occurs. Figure 10 is a high magnification of the notch area in the one-week specimen, showing the presence of the epithelial attachment above the notch area. The process of new bone formation does not appear to last very long as the normal orientation of the traseptal fibers is seen to occur in the eight week period, and only a small amount of bone regeneration and connective tissue reattachment is present in the sixteen-week period. (Fig. 6).

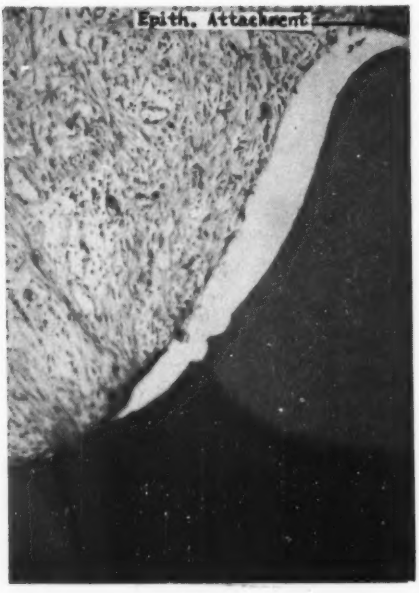

Fig. 10

DISCUSSION

The area which was chosen for the creation of intrabony pocket proved not to be very suitable, for the interdental gingiva of dogs does not have the protection afforded by a contact point as in the human. Moreover, the retention of a dressing or a suture is difficult. However, the histological result demonstrates that osteogenesis can be stimulated by the presence of autogenous, cancellous bone graft in surgically created pathological intrabony pockets in dogs. New spicules grow from the parent bone and join the newly forming bone on the surfaces of the graft to form a new bony trabeculae into which the graft is incorporated. In spite of the fact that some wounds remained open and severe postoperative inflammation occurred, osteogenesis still took place provided that the blood clot remained in the pocket. This suggests the importance of the graft in stimulating or protecting new bone formation, for the amount of new bone formed in the pocket without the graft on the control side was much less at the end of sixteen weeks.

The early experimental specimens always show the arrangement of rapid tissue formation. The arrangement of the connective tissue fibers and capillaries form a bizarre pattern, consisting of numerous blood vessels of different size apparently growing in different directions supported by connective tissue cells and fibers in the process of formation. This pattern gradually assumes order as some of these vessels disappear leaving those in the direction of growth to increase in size. The supporting connective tissue follows the same direction, fiber bundles and finally bone trabeculae being formed.

In the latter experimental specimens, this definite pattern becomes less and less obvious, and finally the upper surfaces of the bone become smooth. The collagenous fiber bundles above it become thicker and arrange themselves horizontally in the mesiodistal direction. This appearance suggests the end of the period of active bone formation. The question of the survival of the osteocytes in the graft is controversial. Most investigators believe that all or most of the osteocytes of the autogenous bone graft die after transplantation. In the present series, only a few osteocytes are found in the lacunae of the graft, and these are in the one week specimens. All lacunae after this period appear empty.

In an area where osteoblasts abound, such as in a healing wound involving bone, a graft may give physical assistance to new bone formation in two important ways; by providing a surface on which the inherent osteoblasts may readily lay down new bone; and by protecting the trabeculae of bone growing from the parent bone from trauma or infection. When a graft is inserted into tissues which contain no bone, formation of bone on the surface of a non-vital preserved graft would suggest that cells of the connective tissue had become differentiated.

It is generally believed that the success

of connective tissue reattachment to the surface of a root of a tooth depends on the absence of epithelial proliferation along the root surface. The connective tissue reattachment can occur only up to the lowest level of the epithelial attachment. In this study, it is interesting to note that inflammation and epithelial proliferation usually occur together and approximately to the same level.

As the epithelial attachment proliferates apically as far as the inflammation extends, it would seem probable that bone regeneration and connective tissue reattachment can only be achieved under favorable conditions. With the process of inflammation, the regenerative process is adversely affected, and the epithelium proliferates apically only to perform its function of covering denuded surfaces.

The suggestions of Carranza[18] and Cross[10] regarding the administration of antibiotics in subgingival curettage for reattachment and bone graft procedures are rationale in that the inflammation arising from bacterial activity will be prevented. It should also be noted that, in spite of extensive downgrowth of the epithelial attachment on the control side, the gingival crevices in the later experimental specimens are always of normal depth.

An almost complete reattachment with bone regeneration which occurred in a case of bifurcation involvement is interesting. One reason for this is possibly the fact that the bifurcation area in the dog is well protected from external injuries. Although the use of bone grafts in bifurcation involvement still needs further investigation, the clinical application in a well-chosen case may be worthwhile.

It is generally accepted that autogenous cancellous bone gives the best result as a graft. However, it has one disadvantage. In order to obtain the graft, an additional operation at the donor site is necessary and this, in turn, gives more post-operative discomfort to the patient. But recently, it is possible that the anorganic graft could be used instead of autogenous bone in the treatment of intrabony pocket and bifurcation involvement with less discomfort to the patient.

CONCLUSIONS

1. Bone regeneration and connective tissue reattachment to the tooth surface can be induced in surgically-created pathological intrabony pockets by the insertion of autogenous cancellous bone grafts under favorable circumstances.

2. Osteogenesis may take place on both the graft and the parent bone.

3. The amount of regeneration and reattachment appears to be in reverse proportion to the severity and duration of inflammation.

4. The mechanism of the "take" of the graft in the intrabony pocket appears to be the same as reported in other bony areas of the body.

5. Bone graft may be successful even when the bifurcation is involved.

SUMMARY

A series of experiments was performed in dogs to study the histological appearances of bone grafts in intrabony pockets after varying periods of time and to determine whether healing takes place by reattachment or adaptation. The methods of the creation of intrabony pockets and the bone grafting operation are described. The clinical and roentgenological observations are correlated with the histological findings. The results show that the intrabony pocket may be eliminated by the induction of osteogenesis by the insertion of autogenous cancellous bone grafts. Healing takes place by reattachment. In the absence of the grafts, the area heals by adaptation of epithelial attachment to the root surface, with a very small amount of new bone formation.

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WESTERN SOCIETY OF PERIODONTOLOGY

The annual spring meeting of the Western Society of Periodontology is to be held at the Tropicana Hotel in Las Vegas, Nevada on March 15-17, 1959. The headline speaker will be Dr. Saul Schluger who is head of the postgraduate division of Washington University, as well as a member of the postgraduate faculty of the University of Southern California. His lecture will be titled "A Critical Evaluation of Periodontal Techniques."

Fee including room and meals, $65.00 for participating dentists and hygienists, and $45.00 for wives.

For information contact Emery Johnson, D.D.S., 250 East 7th Street, Upland, California, or mail reservations direct to the Tropicana Hotel in Las Vegas.

Morphology of Submarginal Calculus

by FRANK G. EVERETT, M.S., D.M.D., M.D.[1] AND GORDON R. POTTER, D.M.D.,[2]
Portland, Oregon

SUBMARGINAL calculus is a hard, dark deposit found as an incrustation on the tooth surface generally within a periodontal pocket. This heavily infected material, lying closely adjacent but outside the epithelial covering of the body, constantly irritates the mucosa. This is similar to the effect of an infected sliver under the skin, but actually worse, because the sliver is within the body and subject to the action of antibodies, phagocytosis, and other defense mechanisms of the body.[1] Calculus, on the other hand, remains on the outside of the adjacent gingiva which reacts to the friction of the infected substance with the formation of an infected decubital ulcer.[2]

Root planing is unquestionably one of the most important dental operations. In the presence of periodontal pockets, it is sometimes difficult even for the skilled operator to divest the tooth completely of all deposits of submarginal calculus. In order, therefore, to ease the job of removing this material, it is necessary to learn more about its morphology.[3, 4, 5]

METHODS

1,132 teeth were extracted from three hundred ninety-six patients suffering from advanced, chronic destructive periodontal disease. During extraction, large or small pieces of calculus were sometimes involuntarily lost from the tooth surface. After extraction the tooth was cleaned of soft materia alba by the use of a multi-tufted, soft-filamented nylon toothbrush. The deposits were then studied macroscopically, under a magnifying glass (3x), and under a dissecting microscope (6x).

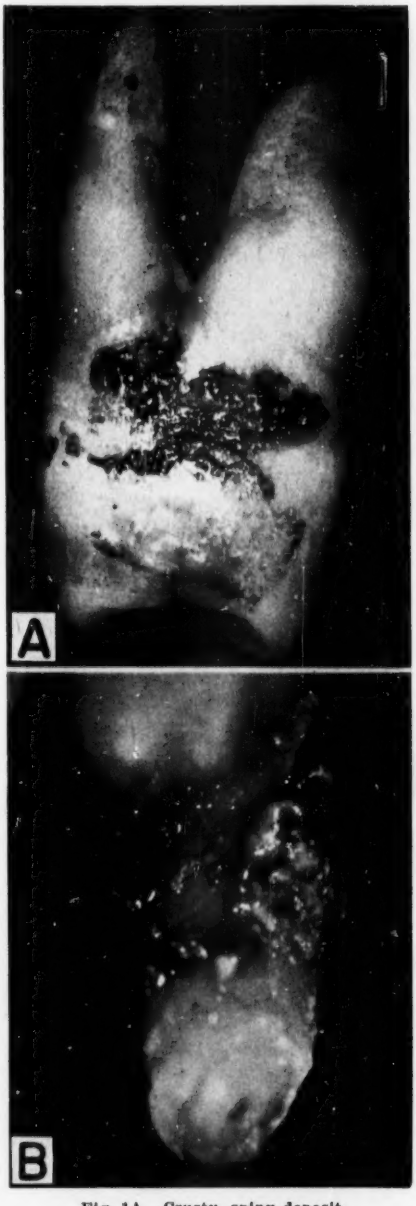

Fig. 1A. Crusty, spiny deposit.
Fig. 1B. Nodular deposit.

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Fig. 2A. Ring-like deposit.
Fig. 2B. Ledge-type deposit.

Fig. 3. Veneer-type of submarginal calculus.

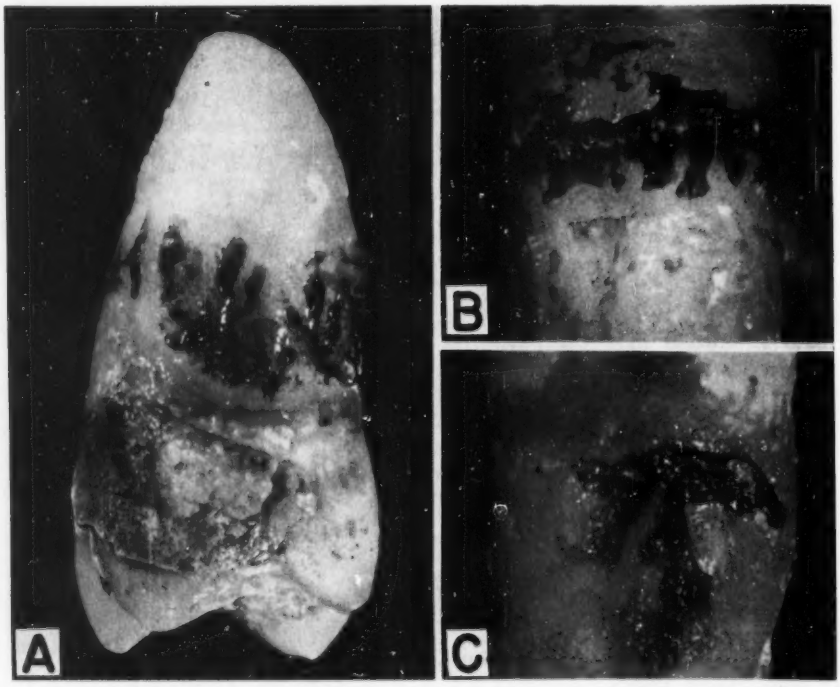

Fig. 4A. Finger-like formation. B. Finger-like formation originating from calculus-ring.
C. Fern-like formation.

Fig. 5A. (a) Individual calculus spots. (b) Veneer deposit. 5B. Individual calculus spots.

RESULTS

The following varieties in the deposition of submarginal calculus could be found in all teeth in the following frequency:

(1) Crusty, spiny or nodular deposits (Fig. 1) 706 cases.

(2) Ledge or ring formation (Fig. 2) 664 cases.

(3) Thin, smooth veneers (Fig. 3) 75 cases.

(4) Finger- and fern-like formations (Fig. 4) 216 cases.

(5) Individual calculus islands or spots (Fig. 5) 52 cases.

(6) Supramarginal upon submarginal deposits (Fig. 6) 6 cases.

It will be noted that the total number of deposits observed exceeds the number of teeth examined, because on one and the same tooth different types of deposits could be observed often.

DISCUSSION

The first three[4] and the sixth[5] of the mentioned six varieties have been previously described. The fourth variety (Fig. 4) finger- or fern-like formations, poses an interesting problem as far as the configuration of the bottom of the pocket is concerned. If, as we assume, deposits always extend to the bottom of the pocket,[3] it is understandable how in the removal of submarginal calculus the epithelial attachment —or at least the finger-like extensions of it that interdigitate with the fingers of calculus—would be often removed in the operation of removal (root planing).[6] It is also interesting that the fingers or fern-leaves in this variety of calculus formation are generally oriented in the long axis of the tooth root.

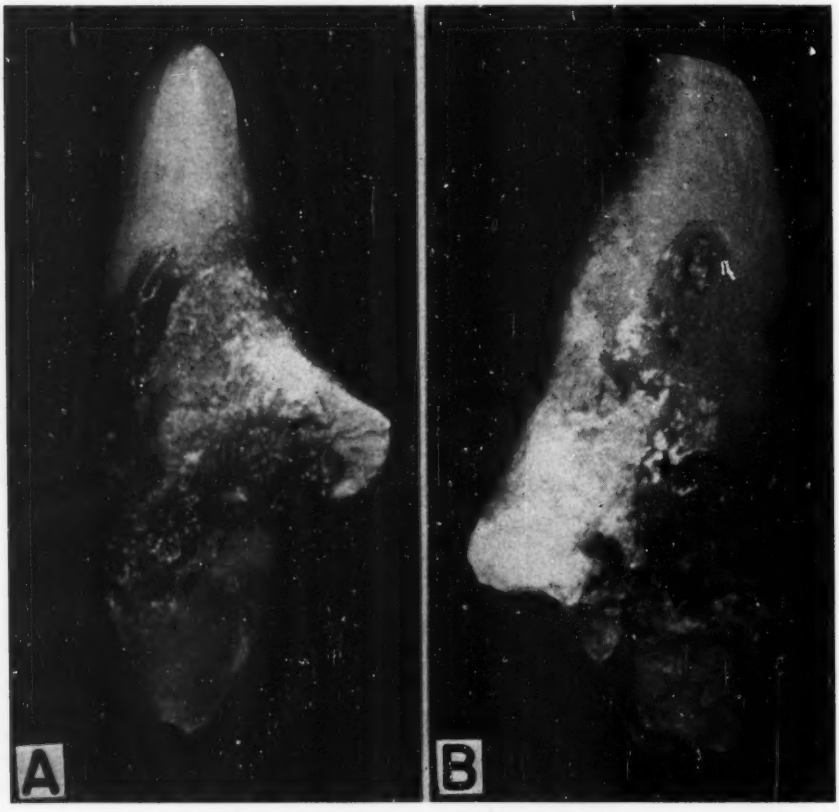

Fig. 6. Supramarginal on submarginal calculus.

Calculus spots or islands (Fig. 5) may be centers of calcification for the material deposited on the root surface. This group thus would represent an earlier stage of crusty type, veneer type, or fern type of formation of submarginal calculus.

The sixth variety was noted only six times. It was seen when submarginal calculus had become exposed into the oral cavity by shrinkage of the gingiva, and when supramarginal calculus had deposited itself on it. This latter observation sheds an interesting light on the question of similarity or dissimilarity of these two concretions. It seems this finding would be much more frequent if supra- and submarginal deposits were of an essentially identical nature.

SUMMARY

Certain variations in the morphology of submarginal calculus are noted. Six forms of deposition are described.

Figures 1b, 2a, 4b and c have been published as illustrations 52 a,b,c and d in Orban B., Wentz, F. M., Everett, F. G., and Grant, D. A., Periodontics, St. Louis, C. V. Mosby, 1958.

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Book Review . . . .

Dentistry for Children. John C. Brauer, William W. Demeritt, L. B. Higley, Roy L. Lindahl, Maury Massler, Isaac Schour. 4th Edition. 492 pages, with 274 Figures. *McGraw-Hill Co., 1958, New York. Price $11.00.*

The reader will find in the Fourth Edition much new material, including a comprehensive review of the current literature dealing with population trends, dental needs and future demands for dental services for the rapidly growing young population of our country.

The areas concerned with practice management and psychological development of the child have been enlarged and contain practical information to cover most situations in the dental office and afford the reader a guide for continued investigation of specialized problems.

The chapters dealing with the development of the teeth embody basic research of many authors and form a sound basis for an understanding of abnormal situations and developmental anomalies and conditions which daily challenge the one who provides dental services for children.

There is no question that dental caries is the number one problem for most dental practitioners. The chapter on dental caries in the growing child is excellent. The section includes current theories relative to etiology of dental caries, with special emphasis on rampant caries. A practical clinical approach to the caries problem is offered, the rationale of which is easy to follow and fits ideally into a modern practice of dentistry.

Diagnostic problems related to gingival and oral soft tissues are given adequate consideration, as in previous editions, and a review of oral changes resulting from nutritional deficiencies cover most of the commonly observed conditions.

Of particular interest to the general practitioner will be the section on technical procedures. This provides a sound basis for operative dentistry for children, the management of pulpally involved teeth, the construction of space maintainers and the special problem of treating traumatic injuries to the teeth.

The Fourth Edition of "Dentistry for Children" is designed as a text for students of dentistry on the undergraduate and graduate levels; however, because of the practical clinical approach by the authors, it will be a valuable addition to the library of the general practitioner, as well as the specialist. *Ralph E. McDonald*

Biochemical and Metabolic Determinants of Experimental Periodontal Disease

by PHILIP PERSON, D.D.S., PH.D.,† *Brooklyn, N. Y.*

PROBLEMS of periodontal disease have rarely been studied in the light of modern biochemical knowledge. From a biochemical perspective, calculus, microbial agents, faulty occlusion, inadequate intake of essential nutrients, and other factors, which contribute to the onset of periodontal disease, do so to the extent that they adversely affect metabolic and physiological processes occurring within the involved tissues. Consequently, whether etiology be primarily "local" and intraoral, or "systemic," or both as is often the case, a most important feature of periodontal disease is disturbance in the metabolism of the affected tissues. Indeed, it is in the physiology and biochemistry of the periodontal tissues that the real meaning of the symptoms of periodontal tissues is hidden. We are faced, then, with the necessity for understanding the role of cell and tissue metabolism as possible determinants of the onset and course of periodontal disease, whatever the exciting causes may be.

In this paper, we will examine within a biochemical frame of reference some of the problems of periodontal disease with special emphasis on microbial agents. It is known that the onset and course of microbial disease may be influenced by host metabolism.[1] As example, the microorganisms responsible for tuberculosis and herpes simplex are almost ubiquitous in urban human populations but their invasion and spread into tissues and subsequent disease production is very much dependent upon host biochemical and metabolic processes.[1,2,3] We have long been aware that the microorganisms seen in necrotic gingivitis are nearly always present in the mouth, yet the disease is not until "tissue resistance is lowered" and organisms invade and proliferate. The role metabolic alterations in diabetes mellitus as determinants of the onset and course of periodontal disease is also well known to all dentists, and may be mentioned.

Rene Dubos, in a recent monograph, has crystallized thoughts such as the above as follows: "Most human beings, indeed probably all living things, carry throughout life a variety of microbial agents potentially pathogenic for them. Under most conditions, these pathogens do not manifest their presence by either symptoms or lesions; only when something happens which upsets the equilibrium between host and parasite does infection evolve into disease. In other words, infection is in many cases the normal state; it is only disease which is abnormal."[4] Considering the microbial aspects of periodontal disease, the nature of the spontaneous occurrences and remissions suggests that we are dealing, not with immunological phenomena per se but as Dubos had carefully emphasized in discussing similar phenomena, with "Situations where physiological-biochemical disturbances are far more important than immunological factors in accounting for the difference between infection and disease. . . ."[5]

Although the management of microbial disease of the periodontal structures has improved considerably in recent years,[6] our knowledge and understanding of host-pathogen interrelationships have not grown in a comparable fashion. The need for in-

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creased knowledge in this newly developing field, i.e., study of the interrelationship between host and microbial metabolism as determinants of clinical disease; and the broad implications inherent in such studies for problems of clinical oral and periodontal disease, in general, have motivated us, in the researches to be reported herein, to investigate host metabolism in relation to the transmittal of experimental periodontal disease. Data from these investigations will be presented indicating that biochemical and metabolic processes within a host may act as determinants of the onset and course of experimental periodontal disease.

RATIONALE OF EXPERIMENTS

Transmission of microbial disease from human mouths to animal mouths has proven difficult in the past.[7,8,9] As a result, most studies dealing with microbial oral disease, including involvements of the periodontium, have dealt with attempts to identify specific causative organisms. Experimental transmission to animals of the lesions of human microbial oral disease has been effected primarily in non-oral tissues (i.e., the groin area), which seem to have yielded better results. Recently, in such studies, Macdonald and his associates working with human gingival crevice scrapings from diseased mouths, were able to establish the identity of some of the organisms causally related to experimental fusospirochetal-type infections.[10,11]

In the present studies, the possibility of transmitting infectious disease to the *periodontal tissues* of experimental animals is re-investigated. Since it is known that the flora seen in microbial gingival disease is almost always present in mouths while the disease is not, it was hoped that by altering the metabolism of the experimental animal host, it would prove possible to successfully transmit to their periodontal tissues, microbial infection and disease from human mouths. The approach used was as follows:

The metabolism of guinea-pigs was altered by the injection of known specific metabolic inhibitors, capable of blocking definite phases of intra-cellular metabolism. This was followed by the injection of homogenized scrapings from the gingival crevice area of diseased human mouths into the sub-gingival tissues of the experimental animals.

It must be stressed that in the studies to be described the experimental host was the prime interest, rather than individual members of the complex flora in the human gingival crevice. For this reason, the *entire* gingival crevice complex was used without attempting isolations, separations, or the establishment of pure cultures. By analogy, here is the reason. For many years, workers in soil micro-biology were led astray because they worked *only* with isolated organisms in pure culture. This was recognized by such great soil microbiologists as Winogradsky and Selman Waksman.[12] In 1951, Quastel in a review of "Nitrification in Soil" described the inadequacy of pure cultures for studying certain complex phenomena, as follows: "It is to be expected that in soil, with its complex microflora and its special physicochemical conditions, the kinetics, possibly even the mechanism, of nitrification, will differ materially from what takes place in pure cultures. There can be no question of the great importance of studies of pure cultures, especially for the investigation of intermediate metabolic products which are unstable in soil. But for the study of the process of nitrification in soil, it is essential to study the course of events in the soil itself, and to look upon the data from pure cultures as complementary to those found in the natural medium."[13] This should not be construed as disrespect for pure culture techniques, which, at the appropriate time, will be necessary in our work. Similarly, transmittal of disease to the *periodontal tissues* of experimental animals rather than other tissues has been a goal of this work, for this too must be done if our knowledge of experimental periodontal disease is to advance and contribute to our understanding of human disease.

METABOLIC CONSIDERATIONS
THE CITRIC ACID CYCLE

Nearly all aerobic mammalian tissues, i.e., tissues which require oxygen in order to live, possess a system of intracellular enzymes known as the tri-carboxylic acid or citric acid cycle.[14] Figure 1 illustrates, diagrammatically, the enzymatic events of the citric acid cycle, which is a major source of energy for cell function. It may be seen in Figure 1, that this system is of added importance because it also serves as a terminal oxidizing path for metabolites produced in other metabolic processes, i.e., glycolysis. Some noteworthy features of the cycle are:

1) Its major components are located intracellularly, primarily within the cytoplasmic particulates called mitochondria.

2) It is the specific system responsible for oxidation of key intermediates of carbohydrate metabolism such as acetate, succinate, citrate, and supplies a major portion of the energy required for cell functions. The citric acid cycle generates the CO_2 we give off in our lungs.

3) This system is also a common path for the integration of carbohydrate, fat, and protein metabolism within the cell.

The existence of some of the components of the citric acid cycle in gingival and other oral tissues has been demonstrated by the work of Eichel[15] and Person and Burnett.[16]

Specific inhibitors of definite steps in the sequence of oxidative enzyme reactions in the citric acid cycle are known. Thus, fluoracetate, as shown in Figure 1, specifically inhibits the oxidation of citrate,[17] while malonate specifically inhibits the oxidation of succinate.[18] Either compound, injected into an animal, will bring to a halt the entire aerobic metabolic cycle, resulting in cell injury or death, depending upon concentrations used.[22] This report will deal mainly with the effects of fluoracetate, although malonate was also used.

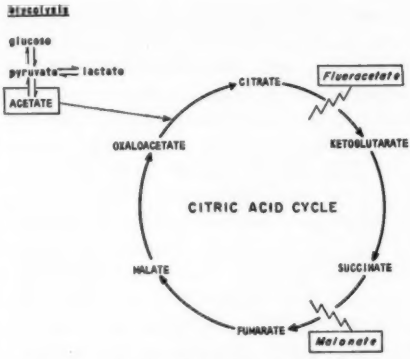

Fig. 1. This diagram illustrates the oxidations of the citric acid cycle, and related events discussed in the text. The jagged lines indicate where fluoracetate and malonate block the oxidations of citrate and succinate, respectively, leading to accumulations of these metabolic intermediates within tissues. On the upper left, the glycolytic metabolic scheme is represented. Glycolysis is an important metabolic path for utilization of sugars in mammals and many microbes. The end product of sugar utilization in glycolysis is the compound, pyruvate, which in its turn may yield lactate or acetate, as shown in the diagram. Acetate may also arise from fatty acid oxidations, and in an activated form combines with oxaloacetate to yield citrate as shown in the long straight arrow. The cycle is thereby renewed and kept in motion. Pyruvate, and some of the other intermediates of the citric acid cycle, may also arise from protein metabolic paths. Thus, the citric acid cycle serves as a terminal, so to speak, for the integration of fat, protein and carbohydrate metabolism within the cell.

Starvation was also used as a means of creating metabolic disturbance. Though not directly applicable to the present experiments, the earlier work of Glickman[19] and Glickman, Morse, and Robinson[20] demonstrated the profound histologic changes in periodontal structures induced by starvation; while the more recent studies of Person and Burnett[16] have shown that the enzymatic oxidation of succinate, in some oral tissues, is decreased as a result of starvation (gingiva not studied).

EXPERIMENTAL

A. Materials and methods.

A total of 347 male guinea-pigs were used in a series of experiments. Animals weighed between 225-400 grams and 600-800 grams. There were no significant findings attributable to age differences. Prior to use, animals were observed for one week

in order to eliminate those with illness. The animals were housed in groups of four to five per cage, and fed Purina Rabbit Chow Checkers and water ad-libitum, supplemented by raw carrots and cabbage twice weekly.

Sodium fluoracetate and sodium malonate were from Bios Labs, Incorporated. Sodium citrate, sodium succinate, and sodium acetate were from Merck & Company. Potassium lactate from Amend Drug Company was used. Solutions were prepared in distilled water and adjusted to neutral pH using a Beckman model G pH meter. Injections of inhibitors and metabolites were made intra-peritoneally. It was found that a fluoracetate dose of 0.25 mg. per kilo body weight produced definite metabolic disturbance, with a minimum of deaths. Malonate, and all other chemicals were prepared as 0.74 molar solutions and injected in 1-2 ml. quantities. Citrate was most toxic, next to fluoracetate, and both these compounds were used efficiently only after some experience in observing their effects. In a group of eight animals, challenged by the above doses of fluoracetate or citrate, one could expect one or two deaths. Animals were challenged with fluoracetate or citrate once or twice at least three to five hours prior to the sub-gingival injections of homogenate. Malonate and succinate were better tolerated and could be injected in a series of four, hourly injections without resulting deaths.

Six patients with moderate to severe marginal gingividites were selected in the dental clinic, and served as sources of gingival crevice material. Three patients were used once, and three were used twice; all in separate experiments involving from 18 to 56 animals per experiment. The patients were all males and were between the ages of 36 to 47. In a typical experiment gingival crevice calculus and debris was removed with scalers. The material was immediately homogenized in saline or other reagents using a ground glass tissue homogenizer. No attempt was made to determine number of organisms, turbidities or nitrogen content of the resulting suspension. The material from a single mouth was usually homogenized in 15 ml. of saline, and 0.1 ml. of this suspension was injected sub-gingivally in the gum covering the bone overlying the lateral aspect of the upper left incisor tooth of experimental animals. Animals were anesthetized by ether during the injection procedure. Glassware and saline were autoclaved, and needles, syringes, and scalers were boiled immediately prior to use. The time interval between beginning of the scaling and sub-gingival injection of the homogenate varied between 15 to 30 minutes.

The following types of controls were employed.

1) As controls for the intra-peritoneal injections of inhibitors and metabolites, intra-peritoneal injections of equal amounts of saline were made.

2) Trauma from the sub-gingival injections, as a factor in the genesis of lesions, was ruled out by the sub-gingival injection of an equal amount of saline on the opposite side of the mouth of the same animal. A further control for this purpose was sub-gingival injection in another animal, of an equal amount of saline on the same side as the homogenate injection.

3) To eliminate the possibility of an inflammatory response to the presence of homogenized calculus and food debris as possible causes of the lesions, suspensions were boiled for ten minutes, cooled, rehomogenized and injected sub-gingivally in control animals. This procedure also eliminated heat-stable toxic compounds as possible causes of lesions.

In one experiment, starvation (complete withholding of food, but water permitted) on the day prior to and following sub-gingival injection of homogenate was used as a means of creating metabolic upset.

A preliminary experiment was also performed in which homogenization of gingival crevice debris and calculus was carried

out in 0.05 molar (final concentrations) of lactate, succinate, citrate respectively, prior to sub-gingival injection.

B. Description and grading of lesions.

Production of lesions by homogenates was usually detectable on the first or second day following sub-gingival injection. The involved gum and its contiguous muco-buccal fold became swollen, edematous, and reddened. In the next two to three days, the swelling localized and definite abscess formation occurred. These abscesses often pointed to the exterior, and frequently were more than one-half centimeter in diameter. Ths pus in these lesions varied in color from yellowish-white to green, was often sanguinous, and varied as to accompanying foul odors. In consistency, the material was at times caseous, at times fluid. Within the next two to three days, the lesion would begin to resolve and go on to uneventful healing. Cellulidites were also seen in which the lesions extended more diffusely throughout the involved gingiva, with abundant production of a sero-sanguinous pus. Figure 2 is a photograph of a typical lesion which had evacuated spontaneously while the animal was being readied for photography. Its swelling was, therefore, reduced but the pointing spots on the gingival surface are easily seen. Lesions were graded arbitrarily, using the following notations:

0 = No change or very slight swelling
1 = Slight edema and swelling
2 = Marked edema and swelling, with redness
3 = Abscess formation, moderate, well delimited, no redness
4 = Abscess formation, severe, diffuse, redness persisting; or cellulitis
H = Healed

C. Results.

1. Effects of sub-gingival saline injections in *non-inhibitor-treated* and in *inhibitor-treated* guinea-pigs.

In four separate experiments, a total of 49 control animals received sub-gingival injections of 0.1 ml. saline. In no case was a lesion ever produced by such an injection.

Fig. 2. A photograph illustrating the appearance of a gingival abscess following spontaneous evacuation of its contents. The lesion was originally about 5 mm. in diameter, and discharged pus a few moments before the photograph was made. The arrow points to the ulcerations through which pus exited.

The wheal raised by the saline disappeared within a few hours. On the following day, the site of tissue penetration by the needle was rarely visible. These observations which were made in *non-inhibitor-treated* and *fluoracetate-treated* animals demonstrate that the trauma, per se, of the injection was incapable of producing lesions, and that the possible introduction of contaminants during the transfers and injections could not have been a factor in the production of lesions.

2. Effect of sub-gingival injections of homogenate in *non-inhibitor-treated* guinea-pigs.

In four separate experiments, a total of 28 *non-inhibitor-treated* animals received sub-gingival injections of homogenate from four different patients, as part of the control procedures in the experiments. The results are summarized in table I, from which it may be seen that homogenate from two patients De L, and B, produced virtually no lesions (1 animal of 17 in-

TABLE I

Production of lesions by sub-gingival injection of homogenate in *non-inhibitor-treated* guinea-pigs

Source of homogenate: Patient	No. of animals injected	No. of animals with lesions	Range of severity of lesions
K	6	6	1-4
De L	8	1	1
Di S	5	3	1-3
B	9	0	0

jected had a grade 1 lesion) while homogenates from the other two patients, K and Di S, produced lesions of greater severity in 9 out of 11 injected animals. For the present, these results indicate that under the conditions of these experiments, the flora from some mouths were capable of producing lesions, while the flora from other mouths were not. Since the total amounts of calculus and debris removed from each mouth were roughly of the same magnitude, this points to qualitative and possible quantitative differences in the flora of these individuals as responsible factors for the differences in lesion production. These aspects of the problem will be studied in greater detail in future experiments.

3. Effect of boiling homogenate on its ability to produce lesions in guinea-pigs.

Two patients whose homogenized gingival crevice scrapings had previously been shown capable of producing lesions were recalled. The purpose of the experiment was to determine whether,

a) irritation by homogenized calculus and debris, and

b) the presence of toxic substances in the homogenates may have been responsible for the production of lesions. No inhibitors were used. Results are summarized in Table II. It may be seen from Table II that boiling homogenates for ten minutes virtually eliminated their capacity to produce lesions, 2 out of 13 animals developing lesions of grade 2, which did not progress further. The same homogenates, not subjected to boiling, produced more severe lesions in 15 out of 15 animals. The latter lesions increased in severity. These data demonstrated that homogenized calculus, food debris, and heat-stable toxic compounds could be eliminated as possible causes of gingival lesion production in these experiments. It is likely, therefore, that visible organisms in the homogenates produced the lesions, although some possible participation by *heat-labile* toxic substances remains to be ruled out.

4. Effects of metabolic disturbances upon lesion production in guinea-pigs.

a) Treatment with fluoracetate prior to injection of homogenate; comparison with effects of citrate and starvation.

The following experiment was one of three performed to determine the effects of prior treatment of animals with fluoracetate on lesion production. Since the net metabolic effect of fluoracetate is a block in citrate oxidation with an accompanying accumulation or piling-up of citrate in the affected tissues, a group of citrate-treated animals was included in this experiment. Also, for purposes of comparison, another type of metabolic disturbance, that produced by starvation, was included. The citrate-treated and fluoracetate-treated animals received two i.p. injections, five hours apart, prior to homogenate injection. The group of starved animals and a fourth control group, received saline injections, i.p., at the same time that the fluoracetate and citrate was administered. In Table III, the initial results of these procedures on lesion production are shown. The experience of the saline controls as seen in Table III showed once more than gingival lesions were produced in the absence of specifically induced metabolic disturbance. Thus on the second day following subgingival homogenate injections, three of the five saline-treated animals had grade

TABLE II

Effects of boiling for 10 minutes upon homogenates ability to produce lesions

Source of homogenate: Patient	Nature of homogenate	No. of animals used	Results following injections
K	Boiled	5	Only one animal developed a lesion, grade 2, on second day. Did not progress beyond this stage. All others negative.
	Not Boiled	8	All animals had grade 2 lesions on second day. Progressed to grade 3 and 4 by third and fourth day.
G	Boiled	8	Only one animal developed grade 2 lesion on fourth day. All others negative.
	Not Boiled	7	Six animals developed grade 2 lesions on second day; one animal developed grade 4 lesion.

1 lesions. On the eighth day following injection, one grade 1 lesion remained and also a grade 3 lesion that had developed in the interval. Treatment with citrate produced a similar initial picture to that seen in the saline controls on the second post-injection day. On the eighth day, the number of involved animals of the citrate group was greater than that seen initially, since one of the animals listed as negative on the second day developed a lesion subsequently. The contrast between the experience of the starved and fluoracetate-treated groups, and the saline-treated group may be seen in Table III. Both starvation and fluoracetate produced lesions of undoubted greater initial severity; furthermore, the lesions persisted for a longer time in these groups than in the saline-treated controls, as may be seen from an examination of the last column in Table III. Thus, to this point in the experiment, results indicated that starvation and fluoracetate treatment produced definite increases in the severity and duration of lesions. The effect of citrate treatment did not match that of starvation or fluoracetate treatment, possibly because the metabolic disturbance induced by citrate, for reasons to be discussed in more detail later, may be more transitory and reversible. The order of healing of lesions by groups was saline, citrate-treated, starved, fluoracetate-treated; i.e., lesions in fluoracetate-treated animals healed last. There were two deaths in the citrate-treated group, and one each in the starved and fluoracetate-treated groups. For this reason, the number of animals indicated under the date 11/1/56 in the last column of Table III, is less than that under the date 10/27/56. These deaths were caused by the metabolic disturbances induced in the animals based upon close observation of their behavior.

In the second phase of this experiment, all animals that had developed lesions, in all groups, were re-challenged with fluoracetate immediately following the healing of the lesions. This re-challenging with fluoracetate was done on an individual basis. Animals were observed at least twice daily during the subsiding of the lesions and as soon as a given lesion could be rated as healed or almost healed, the animal received fluoracetate i.p. The injections were therefore made at different times for the animals of different groups, and even within the same group. Results are summarized in Table IV. It may be seen in Table IV that recurrence of lesions was provoked by re-challenging with fluoracetate in only the starved and fluoracetate-treated groups. It was our experience that this effect could be elicited only when lesions had just healed. If one waited until the second day following healing, and then challenged

TABLE III

Effects of fluoracetate, citrate, and starvation upon lesion production by sub-gingival homogenate injection

Patient: Di S Date of injection: 10/25/56

Metabolic Treatment	No. of Animals	Severity of lesion production on: 10/27/56	11/1/56
Saline i.p. (controls)	5	0, 0, 1, 1, 1	0, 0, 0, 1, 3
Citrate i.p.	6	0, 0, 0, 1, 1, 1	1, 1, 2, 2
Starved	7	1, 1, 1, 2, 2, 3, 3	1, 1, 1, 2, 2, 2
Fluoracetate i.p.	6	0, 2, 2, 3, 3, 3	1, 2, 2, 3, 3

with fluoracetate, recurrences were not provoked. This could be due to the fact that although lesions might appear to be healed clinically, viable organisms remained, and were still capable of lesion production in the early hours following such healing. However, after a day or two, the numbers of viable organisms were probably too few to respond to the fluoracetate injections. It was also our experience that only one recurrence could be elicited under the conditions of these experiments. In other words, after the fluoracetate-induced flare-up healed, subsequent challenges with fluoracetate never provoked additional recurrences. The ability of fluoracetate re-challenging to evoke a flare-up of initially healed lesions, was confirmed in two other separate experiments.

In an experiment in which both malonate and fluoracetate were used as inhibitors, the effects of malonate on lesion production were not as marked as those seen with fluoracetate. In the experiment cited, subsequent re-challenging of malonate-treated animals with fluoracetate did not produce lesion recurrences; while such re-challenging of the initially fluoracetate-treated group did produce recurrences.

These experiments demonstrated that fluoracetate-treatment was capable of increasing the severity of lesions produced by sub-gingival injections of homogenates, when compared with lesions produced by injections of the same homogenates in saline-treated animals. In the experiment summarized in Tables III and IV, starvation in the manner described also increased the severity of lesion production. Treatment of animals with fluoracetate or by starvation, also permitted subsequent induction of flare-ups following healing of lesion, by re-challenging the animal with fluoracetate. Malonate and citrate appeared to be less effective than either fluoracetate or starvation with respect to lesion production. Citrate was used in two experiments; malonate in one.

5. Effects of preparation of homogenates in various metabolites upon lesion production.

Substances such as succinate, citrate, lactate, and acetate, which are intermediary metabolites in mammalian metabolism, are also intermediary metabolites in many bacterial metabolic systems. It is, therefore, possible that one of the ways by which host metabolism influences the growth and invasive abilities of micro-organisms, is through the regulation, via host biochemical processes, of tissue and fluid concentrations of metabolites capable of either activating or inhibiting bacterial growth and

TABLE IV

Effect of re-challenging with fluoracetate following initial healing of gingival lesions, upon the production of recurrences

Group	No. of Animals	No. of animals experiencing recurrence of lesion
Saline, i.p.	5	0
Citrate, i.p.	3	0
Starved	5	3
Fluoracetate, i.p.	5	5

metabolism. A preliminary study was made in an attempt to determine the applicability of this concept to the production of gingival lesions in experimental animals.

In this experiment, gingival crevice scrapings were first homogenized in saline. Three ml. portions of saline homogenate were then immediately re-suspended and re-homogenized in an equal amount of 0.10 molar succinate, citrate, and lactate, respectively. These suspensions were then utilized for sub-gingival inoculation. On the day following inoculation, lesions appeared in the gingivae of each group of animals; those produced by the lactate suspended homogenates were of greater severity than the others.

DISCUSSION

Certain aspects of the present studies merit further consideration. It is felt that immunological considerations played a small role, if any, since re-use of animals in successive experiments is possible (unpublished experiments). If an immunity to the micro-organisms developed, it should not be possible to use animals more than once for the production of lesions, and this we have been able to do.

It is interesting that citrate did not produce as severe lesions as did fluoracetate, although the net effect of fluoracetate is to produce an accumulation of citrate in affected tissues. However, fluoracetate brings the entire metabolic cycle to a halt, while citrate only mimics this effect. Injected citrate can still be metabolized by an active cycle, and this, plus kidney excretion may explain in part the lesser severity of lesions in citrate-treated animals. There are other factors operating, such as effects of the reagents upon the micro-organisms, per se, whirch indicate that the above explanation cannot be the complete story.

Since both fluoracetate and starvation produce widespread metabolic derangements obviously affecting tissues other than the gingivae, consideration must be given to indirect influences upon gingival metabolism and susceptibility. In other words, the experiments reported in this paper do not permit the conclusion that more severe lesion production or induced flare-ups were the result of direct action of fluoracetate or starvation upon gingival tissues *only*. Additional experiments are needed, in which the role of metabolic interrelationships between the various tissue and fluid compartments of the body, and their possible influences upon oral tissue metabolism will be studied.

In the light of the tendency to by-pass gingival and other oral tissues as sites for the production of experimental microbial diseases, the above results acquire added significance. They suggest that the very real difficulties of the past may not have been insurmountable immunological problems, and that attention to details of host metabolic and biochemical processes may offer a new approach to the study of microbial periodontal disease. Although lesions produced in guinea-pigs by the methods described are not identical with the human periodontal lesions which originally harbored the transmitted flora, this should not minimize the significance of the results. Surely, there exist sufficient differences of an anatomic, physiologic, and biochemical nature between human and guinea-pig oral tissues, to make the lack of identity of lesions more reasonable than the demand that they resemble one another in all details. Human tuberculosis and animal tuberculosis differ greatly, and yet the study of animal tuberculosis has been invaluable in the study of the human disease. Avian malaria is not the same as human malaria, yet the study of the avian disease has been of tremendous importance in attacking problems of human malaria. With the proper exercise of judgement and caution, animal experimentation becomes an indispensable pillar of the attack on human disease, notwithstanding the existence of significant differences in the biology of disease in the different organisms.

The present experiments supplement

and may be viewed in the light of other work in which interrelationships between the tricarboxylic acid cycle and microbial disease have been studied. Berry and Mitchell[21] reported that malonate and fluoracetate increased the bacteremias of albino mice infected with Salmonella typhimurium. Survival time of the mice was inversely proportional to the degree of bacteremia, and malonate produced a more severe bacteremia than did fluoracetate. Berry[23] later showed that increased multiplication of salmonella in malonate-treated mice, was not due to detectable differences in the cellular defenses of the mice, since equal amounts of intravenously injected Thorotrast were retained in spleen, liver, and lungs of mice injected with either malonate or saline. Of great interest was the fact that twice as many salmonella were found after growth in a medium prepared from malonate-treated mouse carcasses. This suggested that a metabolic intermediate accumulated in the malonate-treated mice, which enhanced the nutritive properties of the medium for Salmonella typhimurium growth.

Gilfillan, Holtman, and Ross[24] reported that the survival time of chicks infected with Salmonella pullorum was reduced by the administration of fluoracetate, malonate, citrate, and succinate. Approaching the problem from another direction, they also showed that in chicks infected with the salmonella, the functioning of the tricarboxylic acid cycle and other enzymes in liver, was altered. The oxidation of succinate, fumarate, malate, isocitrate, lactate, and cytochrome was significantly influenced by the infectious process.[25]

In addition to specific inhibitors and metabolites of the tricarboxylic acid cycle, other variables have been used to alter host susceptibility to microbial disease. Dubos and Schaedler[26] studied the influence of intraperitoneal injections of pertussis vaccine and of endotoxins of heat-killed Klebsiella pneumoniae and a purified lipopolysaccharide prepared from Salmonella typhosa, upon reversible changes in the susceptibility of mice to bovine tubercle bacillus, Staphylococcus aureus, and Klebsiella pneumoniae. "It was found that mice receiving an infective dose of a virulent culture a few hours after treatment with the endotoxin material, were usually more susceptible to infection than were untreated animals. In contrast, mice infected at a later period proved far more resistant to infection than did untreated animals. The duration of the negative and positive phase of resistance was affected by the amount of endotoxin injected."[26] In a second paper of this series, Schaedler and Dubos[27] investigated the influence of nutritional disturbances upon reversible changes in susceptibility of mice to bacterial infections. It was shown that in fasting animals, and in animals on nitrogen deficient, and fat soluble vitamin deficient diets, the resistance of mice to the organisms mentioned in the previous study was profoundly altered. "Increase in susceptibility to infection could be detected within a few hours to a few days depending upon the nature and intensity of the nutritional disturbance imposed upon the animal. Under the proper conditions, return to a normal state of resistance could also occur within a very short time. There was commonly observed an explosive bacterial multiplication in mice receiving the infective dose shortly after being subjected to nutritional disturbances. Mice rendered more susceptible to infection by being fed deficient diets progressively recovered their normal resistance while being kept on the same inadequate regimens. This adaptation occurred even though the weight of the animals on these regimens remained much lower than the weight of the animals fed a complete diet ad lib. The weight of the animal, at the time of infection, appeared to have little bearing on susceptibility. However, susceptibility consistently increased during the periods when animals were losing weight—whatever the cause of weight loss."[27]

Also pertinent are the studies of Sprunt and Flanigan,[28] who investigated the effects of low protein diets upon the susceptibility

of mice and chickens to influenza virus and to Rous sarcoma virus, respectively. A cyclic susceptibility to virus infection was found. Thus, on a low protein diet there was an initial phase of increased susceptibility. This was followed by a phase of increased *resistance*, and finally by a rapidly increasing susceptibility. It was proposed that these alterations in relative viral susceptibility resulted from metabolic changes within the host during the process of dietary protein depletion. The changes in susceptibility were roughly correlated with the following phenomena:

1) depot fat utilization (increased susceptibility)

2) reserve protein utilization (decreased susceptibility)

3) tissue breakdown subsequent to protein starvation (increased susceptibility)

In section four under presentation of Results, it was mentioned that fluoracetate re-challenging, in the present experiment, produced a flare-up of lesions only once. The work cited above of Schaedler and Dubos,[27] and Sprunt and Flanigan,[28] illustrated the temporal adaptability of organisms to metabolic insult, and the cyclic variations in susceptibility to infection. The possibility, therefore, suggests itself that our guinea-pigs might have adapted significantly to fluoracetate, or that they had entered a more resistant phase for other reasons.

SUMMARY

Gingival lesions may be produced in guinea-pigs by injection of homogenized gingival crevice scrapings from human mouths. When a major energy-supplying metabolic process was blocked, or when animals were starved, there resulted an increase in the severity and duration of such lesions. When fluoracetate-treated or starved guinea-pigs in whom gingival lesions had healed were immediately re-challenged with fluoracetate a flare-up of the lesions resulted. Preliminary experiments indicated that the ability of human gingival crevice flora to produce experimental lesions could also be influenced by homogenization and suspension of gingival scrapings in metabolites common to both host and microbe, such as lactate.

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Book Review . . . .

Accepted Dental Remedies 1959. Published under the auspices of the Council on Dental Therapeutics and the Division of Chemistry of the American Dental Association. 1959 Edition. 224 Pages. Price $3.00.

Accepted Dental Remedies 1959, which bears the insignia honoring the Centennial of the American Dental Association, represents a milestone marking the 25th year in which this publication has been available to the members of the profession. Approximately a quarter of a million copies of Accepted Dental Remedies have been printed during this period.

The new edition contains expanded sections on dental therapeutics. The monographs have been reviewed by the Council and its consultants and have been rewritten in the light of current scientific information. The reorganized format, which made last year's edition so popular, has been retained with minor additional improvements. A discussion of the prescription use of fluoride preparations is included in the chapter on Fluoride Compounds.

As in earlier editions, Accepted Dental Remedies 1959 includes information concerning drugs of recognized value in dentistry, drugs of uncertain status more recently proposed for use by the dentist, and some drugs once employed extensively but now generally regarded as obsolete. Only brands of drugs which the Council has classified in Group A are included in the book.

The Interproximal Stimulator—An Adjunct in Periodontal Therapy

by SIDNEY SORRIN, D.D.S.,* JACK L. THALLER, D.D.S.,** *New York, N. Y.*

THE maintenance of the health of the gingival and interdental tissues accomplished by periodontal therapy is of paramount importance. By means of proper toothbrush massage the following may be accomplished:[1]

1. Establishment of a firm gingival tissue resistant to disease.

2. Stimulation of supporting structures.

3. Keeping teeth clean after they are cleaned by the dentist.

4. Prevention of caries and the formation of calculus.

Due to the inherent limitations of the technique of toothbrush massage, the above results may not be observed in the interdental papillae. The use of a supplementary instrument and technique to fulfill the desired result of health and resistance to disease in the interdental area is of meaningful significance to the dentist and patient. The rubber conical tip as a stimulator becomes a necessary adjunct in the armamentaria of home care.

PURPOSE

Many periodontists[2] have described the advantages of gingival massage. However, the interdental tissue requires special stimulation which is best applied by rubber tip stimulation. This is valuable because of the following:

1. Increases interdental stimulation.

2. Increases interdental circulation.

3. Improves interdental (color) tone.

4. Increases resistance of the interdental gingivae to infection and trauma.

5. Removes materia alba and debris lodged in the interdental embrasure.

6. Helps decrease caries by removal of food.

7. Toughens and tightens the epithelial collar in the interdental embrasure.

8. Reduces proximal sensitivity.

INDICATIONS FOR INTERDENTAL STIMULATION[3]

1. After gingivoplasty and gingivectomy to recontour the topography of the gingival papillae.

2. To reduce enlargement of the interdental tissue due to the use of dilantin, mouth breathing, etc.

3. Where craterizations have formed in the embrasure after repeated attacks of acute necrotizing ulcerative gingivitis.

4. In enlargement of the proximal tissue in full mouth, and partial fixed prosthesis due to over contouring of the veneers, and low soldered contact joints with resultant narrow embrasures which impinge on the gingivae.

5. In enlargement of the interdental tissue due to temporary splints, constructed with acrylic and wire or individually, which may cause impingement of the interdental tissue.

6. For cleansing and stimulating abutments and adjacent pontic areas in fixed prosthesis.

7. For massage of the tissue in manageable bifurcation and trifurcation involvement.

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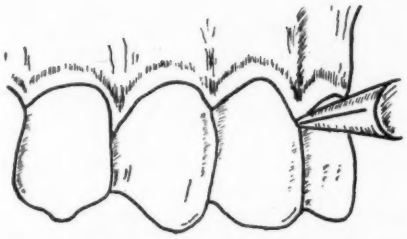

Fig. 1a. The stimulator is inserted in a horizontal fashion into the embrasure as far as possible until the tip of the cone engages the two proximal sides of the upper teeth.

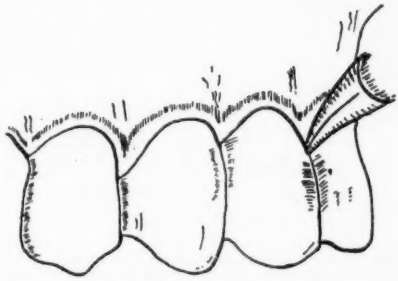

Fig. 1b. The handle is inclined to a 45° angle upward on the upper teeth.

THE INSTRUMENT

A separate distinct instrument to accomplish the technique of interdental stimulation, in preference to the rubber tip placed at the end of the toothbrush, is desirable to secure greater patient cooperation and performance.

The handle of the stimulator should be firm, the shank short, and at right angle to the handle so that the instrument can be inserted with ease in the mouth. The tip of the instrument should be of a hard, flexible, replaceable, latex type rubber that will engage the embrasure with ease.

METHOD

The handle of the stimulator is held firmly, and the stimulator is inserted in a horizontal fashion into the embrasure as far as possible until the tip of the cone engages the two proximal sides of the teeth. Fig. 1a. The handle is now inclined to a 45° angle upward on the upper teeth. Fig. 1b. The purpose of the 45° angle is to guide the interdental tissue in such a manner that it will fill the interdental space and end in a point. If it is employed at a 90° angle it will cause the interdental tissue to be blunted and a space between the teeth will be created. The same procedures are employed in the lower teeth. Fig. 2a, 2b. The point is vibrated with a *gentle* fixed rotary movement to the count of five. Fig. 3. The stimulation produces a massage action with a resultant relaxation of the area. The tip is now withdrawn. Fig. 4a, 4b.

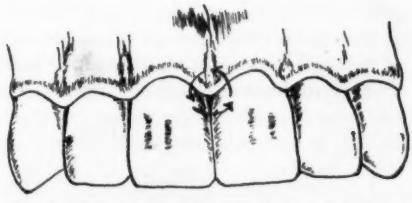

Fig. 3. The rubber tip is vibrated with a gentle fixed rotary movement to the count of five.

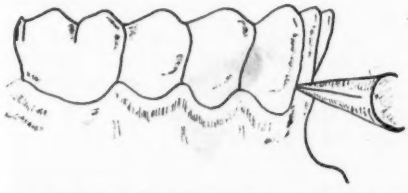

Fig. 2a. The stimulator is inserted in a horizontal fashion into the embrasure as far as possible until the tip of the cone engages the two proximal sides of the lower teeth.

Fig. 2b. The handle is inclined to a 45° angle downward on the lower teeth.

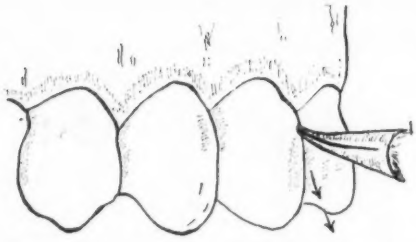

Fig. 4a. The tip is withdrawn downward on the uppers engaging the proximal surfaces of the adjacent teeth.

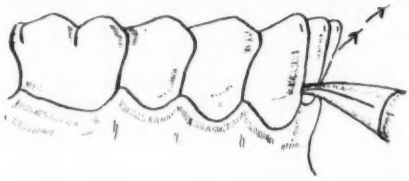

Fig. 4b. The tip is withdrawn upward on the lowers engaging the proximal surfaces of the adjacent teeth.

The adjacent proximal surfaces of the teeth are engaged in removing all the debris, materia alba, and plaques so often present and demonstrable, despite the most diligent and meticulous toothbrushing technique. The procedure is now repeated in all the other interdental areas as demonstrated by the dentist. The patient can be instructed to employ this technique at his leisure while reading, watching T.V., or at any other convenient time.

CONTRA-INDICATIONS

1. Where an intact proximal papillae exists which fills the embrasure supported by intact crestal bone.

2. In any acute inflammatory disease process of the mouth, or where any massage of the oral tissue is contra-indicated, such as blood dyscrasias, neoplasms, etc.

LIMITATIONS

1. Where narrow constricted roots exist with long contact points, especially in the lower anteriors.[4]

2. Where the lingual and buccal interdental surfaces of the posterior teeth are reached with difficulty.

Grateful acknowledgement is made to John W. Lebwith, N.Y.U., 1958, for his art work.

RESULT

The use of the stimulator will result in a firm, hard, interdental gingivae, symbolic of health and freedom from disease.

CONCLUSION

1. A method for the use of the conical, tapered, latex type of tip is demonstrated.

2. The rubber tip is an essential adjunct in mouth care.

3. The dentist must instruct and subsequently check and recheck the use of the stimulator by the patient.

4. The rubber tip is to be used concomitantly with a brush technique for the health of the gingival tissue.

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The Role of the Dental Hygienist in Periodontal Practice

by RICHARD CHACE, D.D.S., *Orlando, Florida*

WITHIN the last few years there has been an unprecedented demand for the services of the American dentist. While formerly only a small percentage of dentists were working at their full capacity, it may be said that now the average well-established dentist is being continually called on to increase his productivity so that he may take care of the patients demanding his services. This increase of productivity must be brought about largely through the intelligent use of ancillary personnel and the refinement of techniques since the increase of chair hours soon results in a drop of personal efficiency. A well trained chair assistant and the laboratory technician have enabled the restorative dentist to tremendously raise his productive capacity. While expert chair assistance is indispensable in some phases of periodontal practice, it is the intelligent use of the dental hygienist that enables the periodontist to meet the increasing patient load. This article may be regarded as suggestive as to the possibility of efficiently utilizing her services in the practice limited to or largely devoted to the practice of periodontia. The limitations of the hygienist as defined by the various state laws have been disregarded. The only points to be considered in this study are whether or not the use of the hygienist will in any way lessen the quality of the service to the patient.

OBJECTIONS TO DENTAL HYGIENIST

There has been a hesitancy among some periodontists to utilize the services of the hygienist. Some claim that their patients would not allow a hygienist to work on them. Some claim that the importance of home care and patient education is not properly emphasized unless the dentist himself teaches it. Others doubt the ability of the hygienist to aid in instrumentation except to a limited degree.

It must be admitted that there are a few patients who will have no part of a hygienist and will insist on the dentist doing all phases of the work. For such persons the hygienist cannot be used but it will be found that these cases are rare, particularly if the patient senses that the hygienist is a trusted partner in the treatment, as indeed she should be. As for the psychology of teaching home care and general patient education, I have found that a patient is much more likely to be impressed by two persons telling him the same thing than he is by only one person's efforts, no matter who that person may be. Even when the patient has great confidence in the doctor, he will often more readily follow the advice of a nurse or hygienist.

The question of technique in instrumentation is a delicate one. It must be admitted that the recent graduate in dental hygiene is poorly prepared to do anything more than remove supra-gingival calculus and stain. She can be trained, however, to use effectively properly designed instruments and to do the preliminary curettage of all but the deepest periodontal pockets. It is true that the periodontist must, at present, give this additional training himself and that it will require some months to develop even the most apt student to a point where her services are really valuable. The required dexterity, however, can be more rapidly attained if it is required that even the most simple calculus removal be done with the fine, subgingival instruments. The exclusive use of such instruments enables the fledgling periodontal assistant to develop the sense of touch so necessary for subgingival work. The writer does not feel that the removal of fine calculus, the planing of the root surface, or the treatment of the soft tissue wall should be done by anyone but a dentist well trained in periodontal procedure. He feels very strongly also that the

dentist who has developed the necessary digital skill for this delicate treatment should not be required to remove the heavy, tenacious deposits in a neglected mouth. Any operator who has had the experience of following a session on such a mouth with an appointment for the definitive curettage of deep, intra-bony pockets will testify that the delicacy of his sense of touch has been lessened by the efforts on the first appointment.

The use of the hygienist for the taking of x-rays depends a good deal on the dentist. There is no reason why the greater part of this phase of practice cannot be assumed by ancillary personnel. For the operator who demands exceptional pictures, the hygienist can learn to make exceptional exposures. She can afford to take several views of certain areas and to retake unsatisfactory pictures. There is nothing about making x-ray exposures that cannot be learned by an intelligent, well trained hygienist who is determined to rise in her profession. The same may be said of intra-oral photographs.

While it is illogical for the hygienist to chart a periodontal case completely, it is quite within her province to make notations on the chart in pencil so that her observations may be of value to the dentist. In order that her capabilities may be fully utilized, she must constantly be watching the mouth for abnormalities or unusual conditions. Preliminary charting of missing teeth, obvious caries, bridgework, etc. may easily be done during prophylaxis, before the final diagnosis is made. Her findings during the prophylactic phase of the recall preventive treatment should also be recorded.

The necessity of a "common language" in recording is necessary since both the dentist and the hygienist are busy on patients and rarely have time to discuss the patient. A glance at the chart is enough for the busy dentist. His orders are, in turn, waiting for the hygienist whenever she sees the patient again.

ACTIVE TREATMENT

The value of the hygienist during active treatment depends largely on the type of case being treated. She is of greatest value in the cleaning up of the neglected mouth. Often several appointments with the hygienist are given for the removal of gross tartar in these cases and for the teaching of adequate home care procedures. Indeed, in the treatment of periodontitis simplex, the home care should be perfected by the patient before the dentist starts work on the case. A certain percentage of patients can never be persuaded to keep their mouths clean, and if the hygienist can weed out such undesirable persons before a great amount of the periodontist's time is wasted, she is rendering a great service. It must be emphasized, however, that during each preliminary appointment, the dentist should spend some time with the patient to observe the progress of the case, and to let the patient know that the important work is yet to come.

The periodontitis complex type of case does not utilize the hygienist to any great extent except for teaching home care procedures. It is recommended that preceding each appointment by the periodontist the hygienist be allowed a few minutes for home care instruction.

The hygienist may be used as a surgical assistant in a gum resection although in a busy office it might be found better to have a chair assistant for this. It is in the repairing and changing of the surgical packs that the dental hygienist saves the dentist many hours each year. The operator must, of course, check the operated area after the pack has been removed and make any necessary medication. Following the healing of a gum resection, the hygienist should have another appointment with the patient for polishing and for home-care checkup.

TREATMENT OF SENSITIVITY

In almost every periodontal practice a major problem of patient comfort is sensitivity of the necks of the teeth. While this

condition is rarely permanent and never dangerous, it is extremely annoying to the patient and operator alike. The treatment of this condition usually consists of burnishing the sensitive area with one of several drugs. Often this burnishing has to be done several times over a period of weeks. In a busy practice, because the dentist knows that the condition is not serious, the patient is too often dismissed before his mouth is completely comfortable. A few extra short appointments with the hygienist for port-polishing will make all the difference between a satisfied, comfortable patient and one whose mouth is never quite comfortable.

MAINTENANCE PHASE OF TREATMENT

Contrary to the practice in many offices, the maintaining of periodontal health after treatment has been completed cannot be turned over to the dental hygienist. All too often the hygienist is asked to assume responsibilities for which she is not qualified. While there are some periodontal patients who need only a prophylaxis to keep their mouths in a state of health, the large majority require close observation by a well trained periodontist in order that possibly recurring lesions be discovered and treated in their incipiency. The dentist who has his hygienist give a prophylaxis and then spends a few moments passing pleasantries with the patient, is not doing his duty to that patient. An ideal procedure is for the dentist to give a short preliminary examination to get a general idea as to tissue tone and home care. A prophylaxis is then given by the hygienist at which time she charts any abnormalities. The dentist then goes over the patient with fine sharp curets and makes any necessary treatment, adjustments of occlusion, and decides how long the patient should wait until the next preventive treatment. For convenience sake it is suggested that certain standard appointments be set up. For instance, the average preventive treatment in this office consists of a fifty minute appointment with the hygienist followed by a ten minute appointment with the dentist. A person with fewer teeth might need only thirty minutes with the hygienist and ten minutes with the dentist.

A disadvantage of any system of recalls is the fact that the dentist sees his patient only when he is at his worse—i.e. when he is in need of a preventive treatment. To give the operator a chance to check the patient occasionally under more favorable circumstances, it is recommended that every two years the hygienist take a full mouth series of roentgenograms and give the patient a prophylaxis. The patient is reappointed in a week so that the dentist can re-chart and re-evaluate the case.

INCIDENTAL DUTIES

There are a number of other duties that the dental hygienist may assume. If caries control measures are a part of office routine, the hygienist is indispensable. Topical application of sodium fluoride, the running of Snyder tests, and analysis of diets are all within the realm of the hygienist. She may also pour study models and construct the acrylic splints that cover the surgical packs.

There is also no reason why the techniques of parenteral medication should not be learned by the dental hygienist. She is the logical person to administer antibiotics or premedication.

And it must be reemphasized that the hygienist is always a teacher. She must teach the importance of home care, the mechanics of home care, and she must assume an important role in the education of the patient in regards to periodontal disease in general.

ECONOMICS

An important part of the consideration of the dental hygienist in the practice of periodontia is the economic one. When the hygienist is employed by a general practitioner she is often paid on a percentage basis. This procedure is not conducive to good periodontia. It is strongly urged that an adequate salary be paid the hygienist plus a percentage of the gross income of

the office. This not only assures the hygienist of extra remuneration when the work is heavy, but it allows the dentist to be the master of the hygienist's time. If the hygienist is to be paid for each service, such as fifteen minutes of toothbrush instruction, an intolerable bookkeeping load is incurred.

The economic import of the dental hygienist on the periodontist is very favorable. The total fee to the patient can be reduced and still the dentist's income will not suffer. By freeing him of considerable drudgery, the hygienist allows the dentist to concentrate on the more difficult phases of periodontia. He will be able to see more patients, and more patients will be able to afford expert periodontal care.

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Book Review . . .

Pediatric Dentistry. M. Michael Cohen and Collaborators, 607 Pages, 395 Illustrations, Including 9 in Color. *The C. V. Mosby Co., St. Louis, 1957. Price $14.75.*

"Pediatric Dentistry" provides a new approach to what is often considered the youngest yet most rapidly growing area, dentistry for children.

The book will serve as excellent reference material for the pedodontic graduate student and the dental intern, in addition to being useful to the undergraduate and dental practitioner. Each chapter is concluded with an excellent bibliography, indicative of the thoroughness with which the material is presented. The reader will be impressed with the excellent illustrations, which assist the busy reader in making a comprehensive review of the material.

Dentistry for the handicapped child is covered more completely than in other pedodontic texts and provides the reader with a stimulus to investigate this relatively new and challenging field.

In many respects, "Pediatric Dentistry" serves as a text of oral pathology in children. The sections devoted to the oral manifestations of systemic disease, and physical and dental developmental anomalies of children are well presented and will be invaluable to the practitioner of dentistry for children and his medical colleagues.

Prophylactic orthodontics and tooth guidance are currently receiving considerable attention by the pedodontist and general practitioner. This text, in addition to containing an excellent review of the subject with an emphasis on diagnostic procedures, outlines treatment designed to prevent or correct many incipient irregularities in the occlusion.

As stated by the author in the preface, "to provide thorough dental care for the child, a knowledge of the basic sciences is essential as related to dental development, sequence of tooth eruption and normal growth and development of the dentofacial complex." The author has followed this philosophy in the development of an excellent text which will be very helpful to the dentist who has as his goal the total dental care for the child.

Ralph E. McDonald

Indices for Prevalence and Incidence of Periodontal Disease

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EPIDEMIOLOGICAL investigations of a disease commonly start with the study of prevalence and incidence of the disease. Prevalence refers to the number of events or defects in an individual or a population at a specified time. Incidence refers to the number of defects that may accrue or accumulate in an individual or a population during a given period of time. When the extent and distribution of the disease has been investigated, the available information is utilized in search for etiological factors and studies of the nature of the disease.

Attempts are always made to gather epidemiological data in numerical systems from which an index can be computed. An index should be quantitative since it must indicate the severity of the disease with reasonable accuracy, and it should be suitable for statistical evaluation.

OBJECTIVES FOR A SCORING SYSTEM OR AN INDEX FOR PERIODONTAL DISEASE

1. To map distribution of the disease (prevalence).
   a. In population groups
   b. Within each dentition
   c. Around each individual tooth (mesial, distal, buccal, lingual)

2. To record the progress and behavior of the disease either by longitudinal studies of the same group or by comparing prevalence studies of various age groups within the same population (incidence).

3. To serve as a basis for evaluation of the role of various etiologic factors in the pathogenesis of periodontal disease.

4. To allow an estimate of total need for periodontal therapy in population groups.

5. To acquire a basis for education of the dental profession, the public, and the governmental authorities regarding the need for attention and treatment of periodontal disease; to estimate future needs for dentists and auxiliary personnel.

6. To serve as a basis for testing and evaluation of various procedures for periodontal treatment.

7. To serve as a basis for evaluation of the effectiveness of various measures in preventing or delaying the loss of teeth from periodontal disease.

8. To serve as a basis for evaluation of measures to prevent periodontal disease.

EVALUATION OF THE LITERATURE

The literature on epidemiology of periodontal disease should be evaluated on the basis of whether or not the previously listed eight objectives have been met adequately. Furthermore, the investigations should be related to the following principles which are fundamental to any type of research.

1. The methods should be described and explained to the extent that the investigation can be duplicated by any scientist in the same field or related fields.

2. The values should be assessed in an objective and documentable way (which is very difficult in clinical research). The ability to reproduce an index or survey system depends mainly upon an exact definition of the criteria for scoring and the ability of the examiner to utilize these criteria under clinical conditions.

3. The conditions for sampling of data should be controlled; the best available scientific tools should be used by properly trained and standardized investigators.

4. Control groups or previously established and acceptable baselines have to be available.

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5. The samples should be representative and of adequate size (size depending mainly upon standard deviation and number of dependent and independent variables).

6. The experimental data should be arranged and processed in such a way that they can be readily analyzed.

When the objectives for a periodontal index and these fundamental principles for research methods are applied to the reported surveys of epidemiology of periodontal disease, it seems that none of the methods listed in the literature even approach the satisfaction of such requirements.

The most common shortcomings of epidemiologic investigations of periodontal disease may be listed as follows:

1. The criteria for scoring have been inadequately described or defined.

2. Inadequate examination methods and tools have been used.

3. Only part of the periodontium has been examined (e.g. P.M.A. index[1]).

4. The assessment of the scoring values has been inadequately documented.

5. The sampling has been inadequate or misleading (for instance, use of anterior teeth exclusively in assessment of prevalence of gingivitis in population groups[1]).

6. The presence or absence of periodontal pockets has not been related to the cementum-enamel junction or any other fixed point on the teeth.

One of the greatest problems in periodontics is the insidious, inconspicuous, and commonly asymptomatic onset and progress of destructive periodontal disease. This problem provides an understandable background for the finding that figures for prevalence of periodontal disease range from 4-5 per cent[2] to 95-100 per cent[3] in similar population groups. The prevalence figures have a tendency to be much higher in surveys conducted by periodontists than reported in surveys by non-periodontists.

The best known periodontal index is Schour and Massler's P.M.A. index.[1,4] Used properly by standardized examiners and applied to the entire dentition, this index seems to be acceptable for the recording of superficial gingivitis. Since it has no provision for recording of periodontal destruction, the P.M.A. index is entirely unsatisfactory for investigation of destructive periodontal disease.

During the last 5-10 years promising attempts have been made to assess clinically and roentgenographically the distribution of insidious as well as advanced periodontal disease in some population groups. Marshall-Day's clinical and radiographic surveys are very comprehensive,[5,6] but his methods are somewhat cumbersome for field studies and the criteria for scoring are not always defined clearly enough to allow duplication by others. The pockets are not related to any fixed points on the teeth. This situation makes it difficult to decide whether or not there is an apparent increase in depth of the crevice because of gingival swelling or the presence of true periodontal pockets associated with the loss of periodontal support. The suggested radiographic technic is inadequate, especially for the maxillary molars (Herulf[7]); therefore, these teeth were excluded from the radiographic score.

McIntosh,[8] Mehta, Grainger, and Williams[9] have included measurements of periodontal pockets in recent investigations. The pockets were not measured or assessed from any fixed points on the teeth, so the periodontal significance of the listed pockets cannot be evaluated. With minor future adjustments their methods seem to have some merit for epidemiological surveys of periodontal disease.

A new periodontal index was suggested recently by Russell.[10] In this index great significance has been given to the presence or absence of periodontal pockets, and the provision has been made to include advanced periodontal disease in the scoring system. Several papers have been published during the last two years utilizing this method.[11,12,13]

For a strictly epidemiological study of variations in distribution of periodontal disease in populations, the Russell index seems to be satisfactory; but evaluated on the basis of the previously listed periodontal objectives, the method has some serious shortcomings.

1. From publications by Russell and coworkers,[10,11,12,13] it seems reasonable to assume that the total prevalence of periodontal disease is grossly underestimated (e.g. 58 per cent with normal periodontium in a group of 601 males aged between 20 and 44 years in Colorado,[10] while Marshall-Day, et al,[6] Mehta et al,[9] and others[14,15] utilizing more exacting procedures for periodontal examination have found only 3 to 15 per cent without periodontal disease in similar age groups). Inadequate examination procedures based on cursory inspection of the gingival tissues without drying off the saliva for evaluation of color, without palpation for density, and without routine probing for pockets is the understandable background for the underestimation of the prevalence.

2. The recording of periodontal pockets by this method is grossly inadequate because of unsuitable tools, lack of routine probing for pockets, and lack of orientation of pockets to the cementum-enamel junction. With superficial inspection a number of pockets will remain unrecognized unless radiographs are available. This error will be most significant in mouths exhibiting good oral hygiene and little overt inflammation.

3. Increase in mobility is not recognized until the tooth is so loose that it cannot be used for function. If mobility should be included as a factor to be recorded, it seems desirable to have some provision for recording it prior to the terminal stage of periodontal disease.

Fairly good comparability of results has been obtained in the Russell method by lowering requirements to accuracy in collecting data, but very few, if any, of the eight previously listed periodontal objectives can be served by this method. It can be argued that a clinical index will never be perfect and that the Russell index has certain desirable features; it is fast and easy to use, it has a fair comparability of results, and it has some provision for the recording of advanced destructive periodontal disease. To some extent the condition around every tooth in the mouth is considered, and finally, it has been used rather extensively, so some basis has been established for comparing periodontal scores from various populations.

Roentgenographic examination is a useful adjunct in surveys of periodontal conditions, but the applied technics need further improvement and standardization (Miller and Seidler,[16] Herulf,[7] Marshall-Day et al,[6,17] Belting et al,[18] and others.[19,20]) These methods, without clinical examination, are totally inadequate for recording of periodontal disease in its various manifestations. Roentgenograms are excellent sources for study of the relative degree of bony destruction around various teeth in the individual dentition, and the use of roentgenograms seemingly has a tendency to increase the over-all score of periodontal disease by the detection of otherwise overlooked areas of periodontal destruction.[10] In field surveys it is difficult to obtain roentgenograms, and it is felt that adequate information can be secured by exacting procedures of clinical examination, including routine probing for periodontal pockets with suitable thin instruments.

Reports on mortality of teeth (Brekhus,[21] Pelton et al,[22]) have provided some indications of the relative order of loss of teeth, and properly sampled it would yield information on the reasons for loss of teeth. This method is, of course, entirely unsatisfactory for studies of morbidity of a disease.

OWN METHOD

The World Health Organization initiated a study of the epidemiology of periodontal disease in India in 1957. As consultant for WHO, I was faced with the

inadequacy of the available systems for recording of periodontal disease. An attempt, therefore, was made to utilize the most valuable features of the previously discussed systems and to add some new principles to compensate for the various listed inadequacies of these systems.

1. It was decided to utilize a selective sampling of teeth for the examination. The most reliable sample is always the entire population, but in most epidemiological studies today, the principles of random or selective sampling are utilized in order to save time and effort. A selective sampling has to be based on a considerable amount of available information in regard to distribution of the disease to be sampled. The pattern of distribution of periodontal disease within the dentition has been studied by numerous investigators using: a) P.M.A. index for gingival manifestations,[23] b) probing and roentgenograms for the detection of formation of pockets and bone loss,[6,7,15,16,17] and c) extraction records for determination of the relative order of lost teeth.[21] Several attempts also have been made to divide the mouth into segments[14] or sections[24] for the purpose of sampling periodontal conditions in the various areas of the mouth. Various selected segments of the mouth also have been used as representatives of the entire dentition.[14,23,25]

Based on these available data and clinical experience from practice and teaching, the following teeth were selected as indicators of the periodontal condition within the dentition:

Tooth number 3 (maxillary right first molar)

Tooth number 9 (maxillary left central incisor)

Tooth number 12 (maxillary left first bicuspid)

Tooth number 19 (mandibular left first molar)

Tooth number 25 (mandibular right central incisor)

Tooth number 28 (mandibular right first bicuspid)

It is felt that a thorough examination of the periodontal status of these six teeth will provide a valid basis for an evaluation of the periodontal condition of the individual.

2. The second important new principle is to record the depth of the crevice or pocket in relation to the cementum-enamel junction. Measuring of the depth of the crevice or pocket has limited value unless the measurements are related to fixed landmarks on the teeth. In order to establish whether or not the periodontal disease has progressed when an eventual re-examination is completed, it is imperative that the available measurements are related to fixed landmarks on the teeth. Particularly when roentgenograms are not available, it is important that the bottom of the pocket is recorded accurately in relation to the cementum-enamel junction. Procedures for examination of pockets, position, and type of periodontal probes are technical problems of utmost importance to the reliability of the recorded data. The available periodontal probes were found to be too thick for probing of narrow pockets, so a new probe had to be constructed ("U. of Mich. #0"). It was attempted to make this probe as thin as possible and to give it the most versatile angulation for universal probing of periodontal pockets. The location of the cementum-enamel junction necessitates removal of calculus if present at this area. It also requires some appropriate instruction and experience to be able to locate this landmark consistently. When the epithelial attachment is entirely on enamel, the cementum-enamel junction cannot always be felt by the probe; but the significant observation then is the absence of permanent periodontal destruction in spite of deep gingival crevices at times.

3. The recording of gingivitis is based on a combination of the P.M.A. index and the Russell index, but the procedures of examination are more detailed than in either

of these methods. The field should be dried to evaluate gingival color. Palpation, probing, and observation should be combined in order to evaluate form, density, and tendency of the gingival tissues to bleed. The presence or absence of gingivitis should be determined by the combined findings of color, form, density, and tendency by the gingival tissues to bleed from palpation and probing. It is extremely easy to overlook early gingivitis; therefore the adoption of these rigid procedures of examination.

4. Calculus and plaques are scored in the search for etiologic factors. Detailed information on the presence of calculus and plaques also is of great importance in the determination of therapeutic needs as well as in evaluation of prophylactic and prevention measures.

5. Attrition is scored as a possible indicator of functional or dysfunctional activity.

6. Mobility is an indication of the functional stress applied on the tooth in relation to its support. To a certain extent mobility is indicative of the degree of periodontal destruction.

7. Lack of contact may be a factor in the etiology of periodontal disease. It may also indicate malposition or drifting of teeth.

The possibility of including malocclusion in the scoring system was considered, but it was not possible to establish acceptable criteria for numerical scoring of malocclusion.

The clinical findings are recorded on the sample sheet (Figure 1). The record also can be printed on a card with the three maxillary teeth on one side and the three mandibular teeth on the other side of the card. The procedures and criteria for the clinical recording follow:

INSTRUCTIONS FOR CLINICAL EXAMINATION

The areas surrounding teeth number 3, 9, 12, 19, 25, 28 should be examined. Dry the field to be examined with cotton. Observe and test the gingival areas around each of the above-mentioned teeth for deviations from health in color, form, density, and bleeding tendency.

Record gingival findings:

G *0* : Absence of inflammation

G *1* : Mild to moderate inflammatory gingival changes not extending all around the tooth

G *2* : Mild to moderately severe gingivitis extending all around the tooth

G *3* : Severe gingivitis characterized by marked redness, tendency to bleed, and ulceration

Record calculus:

C *0* : Absence of calculus

C *1* : Supragingival calculus extending only slightly below the free gingival margin (not more than 1 mm.)

C *2* : Moderate amount of supra and subgingival calculus, or subgingival calculus only

C *3* : An abundance of supra and subgingival calculus

Record pockets:

The distance from the free gingival margin to the cementum-enamel junction and the distance from the free gingival margin to the bottom of the gingival crevice or pocket should be recorded for the mesial, the buccal, the distal, and the lingual aspects of each tooth examined. The interproximal recording should be secured at the buccal aspect of the interproximal contact areas with the probe pointing in the direction of the long axis of the tooth.

A. If the gingival margin is on enamel:

1. Measure from gum margin to cementum-enamel junction and record the measurement on the crown of the schematic tooth. If the epithelial attachment is on the crown and the cementum-enamel junction cannot be felt by the probe, record the depth of the gingival crevice on the crown.
2. Measure from the gingival margin to the bottom of the pocket when the crevice extends apically to the cementum-enamel junction, the measurement should be recorded on the root of the schematic tooth. (The distance from the cementum-enamel junction to the bottom of the pocket can then be found by subtracting measurement number 1 from measurement number 2.)

B. If the gingival margin is on cementum:

1. Measure from the cementum-enamel junction to the gingival margin. Record as minus value on the root of the schematic tooth.
2. Measure from the cementum-enamel junction to the bottom of the gingival crevice. Record value on the root.

Figure I

Name: _____ Address: _____ Case No. _____

Date of Birth: _____ School: _____

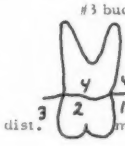
Date of Examination: _____ Examiner: _____

Oral Hygiene Method: _____ Materials: _____ Frequency: _____

Maxilla

#3 buc.

Buccal
G 2
C 1



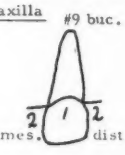
Pocket
A 0
M 0
D 0
Pl 3

Lingual

#3 ling.

#9 buc.

G 0
C 0

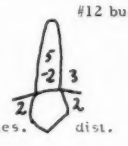


Pocket
A 1
M 0
D 1
Pl 0

#9 ling.

#12 buc.

Buccal
G 1
C 2



Pocket
A 1
M 1
D 0
Pl 2

Lingual

#12 ling.

Mandible

#23 ling.

Lingual
G 2
C 1

Pocket
A 0
M 0
D 0
Pl 2

Buccal

#28 buc.

#25 ling.

G 3
C 2

Pocket
A 1
M 3
D 0
Pl 3

#25 buc.

#19 ling.

Lingual
G 2
C 3

Pocket
A 2
M 1
D 0
Pl 3

Buccal

#19 buc.

Comments:

Record occlusal and incisal attrition:

- **A 0 : No attrition**
- **A 1 : Attrition of enamel only; no exposure of dentin**
- **A 2 : Attrition extends through the enamel into dentin in cuspal and incisal areas, but cuspal pattern has been maintained**
- **A 3 : Extreme attrition; the occlusal surfaces are worn flat and "inverted" cusp pattern is present**

Record mobility:

- **M 0 : Physiologic mobility; firm tooth**
- **M 1 : Slightly increased mobility**
- **M 2 : Definite to considerable increase in mobility, but no impairment of function**
- **M 3 : Extreme mobility; a "loose" tooth that cannot be used for normal function**

Record lack of contact:

- **D 0 : Normal contact; not open**

D *1* : Opening less than 1 mm.

D *2* : Opening between 1 and 3 mm.

D *3* : Opening more than 3 mm.

The side of the tooth that has the greatest interproximal opening is measured. If the adjacent tooth is missing, that area is not measured.

Record plaque after application of disclosing solution:

P *0* : No plaque present

P *1* : Plaque present on some but not on all of the interproximal and gingival surfaces of the tooth

P *2* : Plaque present on all interproximal and gingival surfaces, but covering less than one half of entire clinical crown

P *3* : Plaque extending over all interproximal and gingival surfaces covering more than one half of the entire clinical crown

Only fully erupted teeth should be recorded and missing teeth should not be substituted for their examination. Any effective disclosing solution can be used for location of the bacterial plaques on the teeth. The extent of plaques cannot be evaluated without the use of the disclosing solution, which should be used as the final step of the procedures of examination. It should be applied to all of the teeth to be examined at the same time.

The recorded data can be utilized as a basis for individual index of periodontal disease. Indices for calculus, plaques, mobility, and attrition can be computed. The individual index for periodontal disease is obtained in the following manner:

First, a periodontal score for disease is tabulated for each of the examined teeth. If the gingival crevice in none of the measured areas extends apically to the cementum-enamel junction, the recorded score for gingivitis is the score for periodontal disease for this tooth. If the gingival crevice in any of the four measured areas extends apically to the cementum-enamel junction but not more than 3 millimeters (including 3 mm.) in any area, the tooth is assigned a score for periodontal disease of *4*; the score for gingivitis for that tooth then is disregarded in the final index for periodontal disease. If the gingival crevice in any of the four recorded areas of the tooth extends apically from 3 to 6 millimeters (including 6 mm.) in relation to the cementum-enamel junction, then the tooth is assigned a score for periodontal disease of *5* (the gingivitis score also is disregarded here). Whenever the gingival crevice extends more than 6 millimeters apically to the cementum-enamel junction in any of the measured areas of the tooth, the score of *6* is given as score of periodontal disease for that tooth (again disregarding the gingivitis score).

In this system, as in the Russell index,[10] the numerical values for the scores of periodontal disease are increased in relation to the increasing loss of periodontal support. Whether or not the periodontal support is lost because of periodontitis or atrophy is not considered in this index for periodontal disease. Following the tabulation of the scores for periodontal disease for each of the examined teeth, these scores are added and the sum divided by the number of teeth examined. This division will provide the index for periodontal disease in that individual. Essentially, the method of indexing follows Russell's principles. If some of the teeth which were scheduled to be examined were missing or unerupted so that they could not be examined, the individual scores for each of the examined teeth should be added and divided by the number of teeth examined (if only four teeth were examined, the individual score of periodontal disease is added for each of these four teeth and divided by four to arrive at the index of periodontal disease for that individual.

Indices for calculus, attrition, mobility, contact, and plaques are tabulated in the same manner. For instance, the scores on calculus for each individual tooth examined are added and the sum divided by the number of teeth examined to yield the index on calculus. The scores for plaque for each tooth examined are added and divided by the number of examined teeth, and the index of plaque or oral hygiene is achieved. In the same manner indices for attrition, mobility, and contact can be achieved.

The data then are ready for analysis and statistical evaluation. Interrelation between any of the scored factors can be tested and evaluated statistically.

Distribution and progress of periodontal disease around the individual tooth mesially, buccally, distally, and lingually also can be studied from these records.

The data recorded on the chart shown in Figure 1 can be used to illustrate the procedures of computation. The score for periodontal disease of each tooth from Figure 1 would be as follows:

For tooth number 3, *4*

For tooth number 9, *0*

For tooth number 12, *5*

For tooth number 19, *6*

For tooth number 25, *4*

For tooth number 28, *2*

The sum of these individual scores is *21*, and divided by 6 (the number of teeth examined) would yield an index of periodontal disease of 3.5 for this individual. The sum of the scores on calculus would be 9, and divided by 6 would yield an index on calculus of 1.5. The index on attrition is 0.8; the index for contact is 0.2; the index for plaque is 2.2.

Using but one decimal point in tabulation of individual indices and raising or lowering to the nearest fraction of hundreds, one reduces the decimal; for instance, one divided by six is recorded as 0.2 and two divided by six is recorded as 0.3. For computation of scores on population two decimal places should be used.

SUMMARY

A quantitative method of scoring periodontal disease and related conditions has been described. For each individual it requires a thorough examination of six sample teeth and surrounding tissues. Periodontal pockets are related to the cementum-enamel junction to provide the periodontal significance of the measurements. Various etiologic factors are recorded. All recordings are completed in numerical systems to facilitate indexing and analysis; ambiguous criteria have been avoided; the method is easy to learn, and the comparability of examiners has been as good as can be expected from any clinical periodontal index;[26] minimum equipment is needed; and the method is quickly applied—less than five minutes for each mouth.

An attempt has been made to meet the various requirements for a system of periodontal scoring applicable to epidemiological, clinical, and combined epidemiological and clinical investigations.

A detailed analysis of the clinical tests of this method will be published in a few months through the World Health Organization.

ACKNOWLEDGMENTS

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ALPHA OMEGA FRATERNITY

"Total Periodontal Concept" is the theme of the premiere Sunshine Seminar to be presented by the Alumni of the Greater Miami Chapter of Alpha Omega Fraternity. This initial program will be at the Deauville Hotel, Miami Beach, from January 25 through January 30, 1959. Clinicians include Drs. Morris Amsterdam, Walter Cohen, Don McQueen, Balint Orban and John Pritchard.

Complete information about this program may be obtained by writing Dr. Irving Gordon, 420 Lincoln Road, Miami Beach 39, Florida.

The Periodontal Care Concept—Dentistry's New Horizon

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UNTIL rather recently the dental profession, by and large, steadfastly pursued a course of negative "downhill" dentistry regarding retention of the natural dentition in health for the lifetime of the patient. The vast majority of the profession was willing to believe, and so indoctrinated their patients, that very little really could be done for "weak gums" or so called "pyorrhea" and that eventually natural teeth would be lost and replaced by "nice dentures." All dental procedures that were done, somehow, from earliest childhood, seemed to be predicated on the concept that most adults would wear full dentures in their lifetime. Against this ominous note there was but a handful of clinicians who did not believe this to be so and whose clinical results demonstrated to themselves that it need not be so. But the pressure from within the profession to wilfully discredit and ignore their findings and observations was so great that little headway could be made in changing this mass concept of the profession.

Let us, first, analyze the status of periodontal disease in the United States today from the viewpoint of epidemiology, to determine whether in reality periodontal disease is truly a major oral health problem, or whether we are making "too much to-do" about the status of this problem in the thinking of modern dentistry. The following statistics are only a few from the ever-growing bastion of evidence clearly setting forth the magnitude of this problem with which progressive dentistry is now faced.

Edward F. Allen,[1] in 1944, made a statistical study on the primary causes of tooth extraction. He demonstrated here that caries and periodontal disease accounted for 92.6% of tooth loss. Up to 35 years of age, caries appeared to be the major cause of tooth loss. However, beyond the age of 35, periodontal disease became the major cause of tooth loss. For example, he demonstrated that the average tooth loss per visit by extraction for caries was three teeth per patient per visit; whereas for periodontal disease the incidence was ten teeth lost by extraction per patient per visit—in other words, in the adult, *periodontal disease* is conclusively pinpointed as the cause of mass extraction of teeth and *not tooth decay.*

However, we should here note that periodontal disease is not limited merely to the 35 year old and over age group, since existent knowledge demonstrates to us that in this age group the disease process is mostly chronic, having developed over a period of a number of years, and especially[2] from an early inception. It has been our observation, in doing routine oral examinations of elementary and high school students, that the evidence of inflammatory changes in the gingivae of elementary pupils is great, and that the existence of chronic destructive periodontal disease in high school students is significant. These observations readily correlate with the high existing incidence of already developed periodontal disease in young adults.

This was shown by a most significant study recently carried out by the Metropolitan Life Insurance Company[3] which indicates definitely at least an 80% incidence of periodontal disease in the adult population of 18 years of age and over. To state it more visually, according to this study at least 8 out of 10 of the entire adult population in the United States, 18 years of age and over, can now be expected to have some degree of clinically detectable periodontal disease.

It becomes evident, then, that the periodontal disease problem desperately needs to become the concern of modern dentistry

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for patients of every age group. For if we are to think along lines of rational therapy as regards treatment of any health problem, we can no longer consider it adequate to attempt intervention merely at the later chronic destructive stages. On the contrary, we must make every effort, utilizing all available knowledge, to detect periodontal disease early, and above all, to treat promptly and completely, thus necessitating less complicated treatment regimes in order to assure the gratifying clinical results— which are readily obtainable today.

And yet, if we carry our line of thinking but one step further, we arrive at the ultimate in therapy—and that is, even more important than early treatment—*prevention.* The achievement of this goal necessitates the utilization of every effort to detect any and all conditions capable of producing periodontal disease; to correct these conditions wherever and however possible; and most important of all, to institute clinical regimes designed to permanently contracept the initiation of periodontal disease.

It is truly unfortunate that an all too prevalent notion exists, regarding periodontia as consisting of a conglomeration of treatment procedures concerned with futilly heroic attempts to retain so-called "hopelessly" involved teeth. But the dental profession today, at its higher eschelons certainly, has dispersed this fog of ignorance and distortion and is adopting this new concept for periodontics—a broad concept which Irving Glickman has presented as "The Periodontal Care Concept."[4]

"Periodontal care" represents the comprehensive approach toward maintenance of the health of *all* periodontal tissues to *all* the teeth of *all* patients. In this concept, there should be no differentiation as to a "periodontal patient" and a patient who is not a "periodontal patient." For in reality, the vast majority of patients who are not willingly cooperative in seeking periodontal treatments are patients who have not been properly educated to the understanding of periodontal health and the maintenance of their natural teeth for their lifetime. To continue considering large groups of patients as so-called "poor risk" patients is to admit that, as a profession, we are unwilling to carry out proper patient education programs in our public health pursuits, as well as in our practices. That this is a problem vast and difficult is not denied—but that this is a problem which modern dentistry *must* assume as a responsibility—there can be *no question.*

"The Periodontal Care" concept emphasizes *care* of healthy tissues as well as diseased tissues. In the case of apparently healthy tissue, periodontal care is a prophylactic preventive measure; whereas in the case of diagnosed recognized periodontal disease, the "periodontal care" concept affects itself as the "modus operandi" by which treatment to effect the complete elimination of the disease process and the creation of conditions conducive to the maintenance of complete periodontal health, *indefinitely,* is carried out. These conditions must utilize the vast span of every phase of modern "positive" dentistry: periodontics, including the utilization of "Oral Orthopedic Splinting"[5] wherever indicated, operative, endodontics, and reconstructive prosthesis both fixed and removable—all predicated to effect the maintenance of, or restoration to, an optimum balanced, functioning esthetic masticatory mechanism based on the retained natural dentition.

This overall co-ordinated approach can be very effectively termed "Combined Periodontic-Prosthodontic Therapy."[6] Details of this therapy plan, along with clinical presentations and photographs will be presented in papers now in preparation by this author.

Therefore, the "Periodontal Care Concept" encompasses: 1, The *prevention* of pathologic changes in the supporting tissues; 2, The *treatment* and *return to normal,* or to the *maximum health* possible; and 3, The maintenance of the attained maximum health of the supporting tissues correlated

with the *optimum functioning* of the *retained natural dentition* for the *lifetime* of the patient.

Goldman[7] has since further added to the clarity of this clinical regime by referring to a twofold objective for complete periodontal therapy as "(1) the eradication or arrest of the periodontal lesion with the correction or cure of the deformity created by it, and (2) the alteration in the mouth of the periodontal climate which was presumably an important contributing factor to the periodontal breakdown." This formulation blends quite logically with what has already been said concerning the extension of the "Periodontal Care" thesis into the single Periodontic-Prosthodontic therapy concept.

Thus, it follows that "periodontal care" inculcates a periodontal consciousness into the practice of dentistry.[8] It insists on a need for *eliminating all* potential causes of periodontal disease in *all* patients. It cautions against the creation of conditions which can induce periodontal disease, at *any* stage in the lifetime of the patient. It demands, then, prevention from the start, as well as the earliest possible recognition and *total treatment* of periodontal problems which have already developed.

In essence this represents modern dentistry's "crusade" to predicate itself as truly equal in the health sciences. This concept pinpoints and crystallizes what must, for modern dentistry, become its greatest contribution as a health profession—the "know-how" to maintain the oral masticatory mechanism in optimum health and function based on retained natural dentition for the lifetime of the patient. The "know-how" which every patient is entitled to understand is the *existing obtainable maximum of prevention and therapy.*

This "know-how" demands, on the basis of the evidence of existing clinical results,[9] a complete re-evaluation of such casually accepted negative generalizations in treatment planning as "when in doubt take it out," "don't be a hero," "extract now and save the ridge," etc.

The increased responsibility must be assumed if the patient is not to be deprived of this vastly increased scope of therapy making possible the functional reconstruction of periodontally involved dentitions—through the utilization of such "Combined Periodontic-Prosthodontic Therapy."[10]

Thus, with the application of these concepts in clinical dentistry, we will move into a true "Periodontal Care Era"[11] marked by this new delineation in the health service being made available by an envisioned, forward-seeking dental profession. And, thus, will become a reality the ever increasing hopes of an educated public to retain in health their natural dentition for their lifetime.

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Triamcinolone Acetonide in the Treatment of Recurrent Ulcerative Stomatitis

Preliminary Report

by EDWARD V. ZEGARELLI, D.D.S., M.S., AUSTIN H. KUTSCHER, D.D.S., AND HERBERT F. SILVERS, D.D.S., *New York, New York*

THE treatment of recurrent ulcerative stomatitis has until the era of the corticosteroids been characterized generally by negative findings and failure. In the hands of various investigators, numerous corticosteroids including prednisolone, hydrocortisone, fluorohydrocortisone, and triamcinolone have all been described as useful in controlling the lesions of recurrent ulcerative stomatitis. We have recently reported on a group of patients with recurrent *scarifying* ulcerative stomatitis in whom triamcinolone and prednisolone were found to be markedly effective. In this latter study,[1] triamcinolone was incorporated in a new adhesive vehicle—which vehicle may have contributed in some degree to the favorable results obtained with triamcinolone. Although the individual lesions at the time of active triamcinolone therapy were markedly improved as to both duration and severity, the *recurrence* of lesions was not prevented and the severity of recurring lesions was not decreased.

The beneficial treatment of recurrent ulcerative stomatitis (still, unfortunately, far short of the desired "cure") is reported herewith using a combination of a new corticosteroid, triamcinolone acetonide, incorporated in the previously mentioned adhesive vehicle.

Triamcinolone acetonide is a new synthetic corticosteroid (a derivative of triamcinolone) which has been found to be particularly active upon topical application although it is also a fully active corticosteroid when administered systemically.

Pharmacological data on triamcinolone acetonide: The results of an initial 5-day cotton-pellet test[2] in adrenalectomized rats indicate that triamcinolone acetonide possesses anti-inflammatory activity equal to about 20-35 times that of hydrocortisone acetate, 10 times the activity of prednisolone, and 10 times the activity of triamcinolone alcohol as an anti-inflammatory agent in animals.

Triamcinolone acetonide has been compared to cortisone acetate, hydrocortisone acetate, and triamcinolone alcohol in glucocorticoid activity by the rat liver glycogen assay.[2] In this test, it showed a potency over 90 times that of cortisone, 20 times that of hydrocortisone acetate, and greater than 9 times that of triamcinolone alcohol.

Thus far, triamcinolone acetonide has exhibited no sodium retaining activity in animal experiments;[2] rather, it has produced an excretion of sodium.

Triamcinolone acetonide has been used topically almost exclusively, having been administered systemically primarily to supply pharmacology data in man.

Triamcinolone acetonide was administered[2] orally to 5 patients with rheumatoid arthritis in doses ranging from 1 to 10 milligrams for periods of 1 week to 2 months. All the patients had previously been satisfactorily maintained relatively free from signs and symptoms of the disease with prednisone in doses of 7.5 to 15

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This study was supported in part by a grant from The Squibb Institute for Medical Research, New Brunswick, N. J. Triamcinolone acetonide (Kenalog(R)) in an adhesive vehicle available from E. R. Squibb & Sons as "Orabase" was supplied by The Squibb Institute for Medical Research, New Brunswick, N. J.

milligrams per day. In 2 patients, triamcinolone acetonide was as effective as prednisone in controlling signs and symptoms of the disease (in one patient at a dose level of less than 1 mg. per day), while in the other 3 patients, triamcinolone acetonide was less effective. Except for drowsiness, anorexia, and a loss in weight (4 to 5 pounds) over a period of one month in 1 patient, no other undesirable or untoward effects of the administration of triamcinolone acetonide were reported. No clinical evidence of significant electrolyte disturbance was noted.

Triamcinolone acetonide was administered[2] to 4 children (age 5½ to 9 years) in relatively massive single oral doses (30-50 milligrams). Suppression of 17-ketosteroid excretion (as measured in 24 hour urine samples), as well as suppression of endogenous corticoid production (as measured by both the blue tetrazolium and Porter-Silber reactions) were observed. Maximum suppression of endogenous corticoid production occurred within 12 hours of dosing, and this suppression persisted for as long as 36 hours.

METHODS

Nine patients, aged 9 to 55, all with typical histories and evidences of recurrent ulcerative stomatitis were studied. All patients had been followed through numerous episodes of their recurrent ulcers and through courses of therapy with various agents including placebos and/or often, other topical corticosteroids.

Triamcinolone acetonide in an adhesive vehicle* was applied to lesions by the patient 4 times a day in a 0.1% concentration. Approximately 60-250 mg. of the preparation was to be applied sparingly in a thin film (swelling of the adhesive vehicle occurs shortly after contact with the saliva) after meals and especially before retiring. Thus, about 240-1000 mg. of the preparation including therein .24 mg. to 1 mg. of triamcinolone acetonide was to be applied to a lesion during the course of a full day.

*This vehicle ("Orabase") coats lesions with a desired therapeutic agent over prolonged periods thus: (1) increasing the potential effectiveness of drugs by means of maintaining higher concentrations of the agent at the site of a given lesion; (2) decreasing the total amount of drug which need be applied; (3) increasing the duration over which the material will remain in situ; (4) decreasing the amount of material which, following displacement from the area, would be swallowed and would hence be available for systemic activity; and (5) providing also a mechanically protective layer over the lesion.

RESULTS

When the course of untreated, placebo treated, and corticosteroid treated (other than triamcinolone acetonide) lesions were compared with the course of triamcinolone acetonide treated ulcers in our group of patients, it could be concluded that triamcinolone acetonide in the adhesive vehicle is useful in controlling lesions of recurrent ulcerative stomatitis. This observation was evident in all patients studied in this series. The therapeutic complex of triamcinolone acetonide in the vehicle adhesive is often able to abort lesions at their onset—if applied sufficiently early in the cycle of a given developing ulcer. This is particularly notable if the lesions are treated at the time of prodromal symptoms, i.e. before an actual vesicle or ulcer has formed.

It would seem from study of triamcinolone acetonide treated ulcers that the use of this corticosteroid results in the reduction of the usual duration of an untreated ulcer by approximately one-half. Distressing subjective symptomatology (soreness and pain) and objective evidence of inflammation and ulceration respond rapidly. Overall, both the duration and the severity of lesions are more markedly decreased with triamcinolone acetonide therapy, in our experience, than with other corticosteroids. It is not unusual to be able to effect healing or at least initial signs of epithelialization with loss of symptomatology within three days from the beginning of therapy.

The application of the vehicle-adhesive per se without the incorporation of the

acetonide results in some relief of symptomatology for the duration of the time during which the adhesive remains at the site of the lesion. Upon dissolution of the protectant, the usual pain and symptomatology return at once. There is no alteration in the total duration or overall severity of lesions with the use of such protectant therapy alone, however.

It should also be emphasized that, in common with other corticosteroids, the administration of triamcinolone acetonide does *not* prevent the reappearance of lesions upon discontinuation of therapy nor is there any decrease in the size or severity of the recurrent lesions or in the duration of such recurrent lesions. Also, the remission period between lesions is not significantly increased — if therapy is discontinued promptly upon healing of a given crop of ulcers.

Weakness was noted by two patients upon prolonged although discontinuous therapy with triamcinolone acetonide—an observation not inconsistent with the pharmacology of this drug. No other side effects were noted.

DISCUSSION

The advantages of triamcinolone acetonide concern its improved topical activity rather than any unique systemic action. Hence, its use and its usefulness in general have been limited to topical administration. Nevertheless, triamcinolone acetonide is fully active systemically, and such medication as the patient ultimately swallows is then available for full systemic action. On a weight basis, triamcinolone acetonide is a potent corticosteroid with powerful pituitary suppressant activity—which action can readily be demonstrated at the laboratory level. As with other of the newer corticosteroids including prednisolone and triamcinolone, preliminary studies suggest that triamcinolone acetonide does not serve as adequate replacement therapy for suppressed adrenocortical activity—via pituitary inhibition. The above described muscular weakness probably is related to potassium excretion resulting in potassium depletion — the mechanism of which is now being explored. Hence, the duration of administration and total dosage of this steroid must be watched carefully.

Observation of the characteristics and course of lesions under treatment seem to indicate a beneficial action of triamcinolone acetonide on the basis nearly exclusively of anti-inflammatory action alone. The systemic activity of the ingested corticosteroid may be a factor in the beneficial action of this compound as utilized in this study.

The use of the adhesive vehicle markedly decreases the total quantity of the active drug necessary to induce healing and hence represents an important part of the therapeutic complex. It is imperative that the patient make the maximum use of the adhesive properties of this preparation by applying the triamcinolone acetonide in vehicle adhesive just before retiring—thus permitting steroid contact with the tissues throughout the entire night usually. In order to minimize the total dose required also, it is obvious that the patient should apply the drug at the earliest sign of the onset of a lesion, thus perhaps making possible the abortion of the cycle rather than the treatment of an overt lesion.

SUMMARY

Triamcinolone acetonide, a powerful new synthetic corticosteroid, has been used with marked success in the treatment of episodes of recurrent ulcerative stomatitis in 9 patients. This topically applied corticosteroid, when incorporated in a new adhesive vehicle, has been found to be effective in aborting lesions (if applied promptly following initial prodromal symptomatology) and in obtaining rapid healing or beginning epithelialization of existing ulcers. It should be emphasized that, in common with other corticosteroids, the administration of triamcinolone acetonide does *not* prevent the recurrence of

lesions and does *not* result in any decrease in the size, severity, or duration of recurrent lesions—unless the drug is readministered. The remission period between lesions is *not* increased if therapy is discontinued upon healing of a given crop of ulcers. Thus, triamcinolone therapy should be administered only with the full realization that its action is entirely suppressive and temporary rather than curative.

Since triamcinolone acetonide is a powerful corticosteroid which is fully active systemically, the total dose administered and the duration of therapy must be watched carefully. It is also essential that therapy be of a discontinuous nature and that particular advantage be taken of the adhesive vehicle's ability to maintain a drug at the site of application by applying the therapeutic agent before retiring thus permitting a maximum of triamcinolone acetonide activity throughout the course of the bulk of the night with a minimum of total drug administered.

Further clinical studies of this compound in the adhesive vehicle are indicated in recurrent ulcerative stomatitis as are clinical studies in a variety of other acute and chronic lesions of the oral mucous membranes.

The authors acknowledge with gratitude the aid of Mrs. Florence V. Moore and Miss Joan Graham in coordinating these studies.

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Instrumentation in Ultrasonic Periodontal Therapy

by Sol J. Ewen, D.D.S.,* Paul J. Tascher, D.D.S.,** *New York, N. Y.*

RECIPROCAL action instruments that vibrate at frequencies beyond the audible range have been shown to be valuable in the treatment of the periodontium.[1,2,3] For example, ultrasonic vibrations are able to satisfactorily debride human teeth of surface accretions; existing restorations can be reshaped to physiologic contour; gold margins can be burnished smooth; and periodontal pockets can be flushed with water or medicaments.

This report will present several techniques extending the usefulness of ultrasonic vibrations in periodontal therapy.

INSTRUMENTS FOR SUBGINGIVAL SCALING

In attempts to reach the depths of periodontal pockets one of us (P. J. T.) has previously designed instruments that operate without detrimental effects to the hard and soft tissues.[4] Other investigators, employing heavy, single edged, blunt instru-

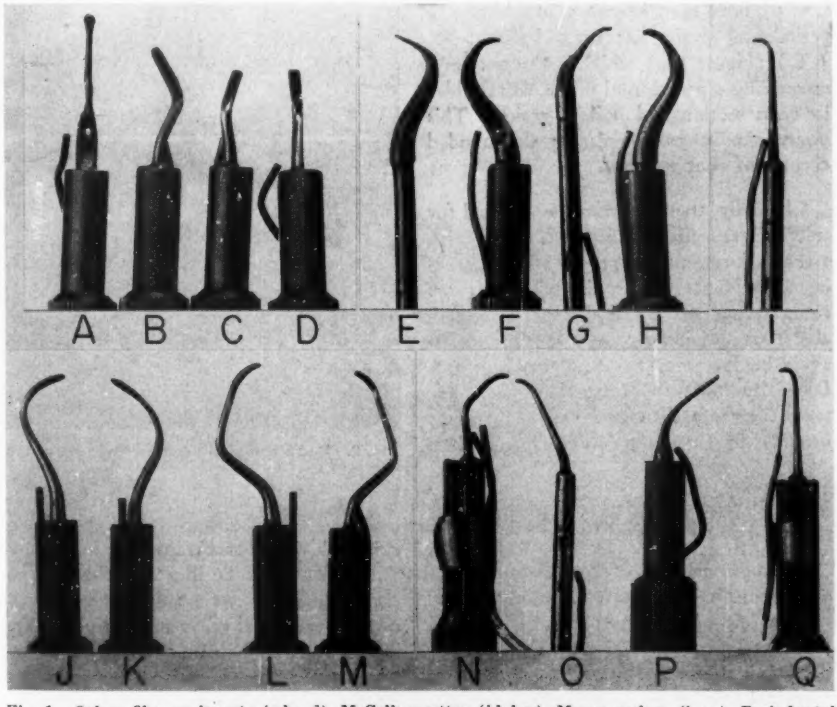

Fig. 1. Orban files on inserts (a,b,c,d). McCall curettes (j,k,l,m). Morse scalers (i,n,o). Periodontal probe and scaler (p). Instruments for contouring restorations (e,f,g,h). Medicament delivery tube (28 gauge) (q).

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ments have shown how efficiently stain and supragingival calculus may be removed.[2] However, since the vibrating tool tip is a *reciprocal action device* moving in a push and pull direction, instruments with two edges should be even more effective for subgingival scaling. In clinical trial this has proven to be the case. These instruments resemble traditional files, curettes and probes which are brazed to magnetostrictive stacks.

For gross subgingival debridement transducer inserts using dulled Orban files (Figure I, a, b, c, d) and McCall curettes Nos. 13, 14, 17, and 18 (Figure I, j, k, l, m) are suggested. For detailed root planing Morse scalers Nos. 0 and 00 (Figures I, i, n, o) have been effective. In addition, a periodontal probe has been designed (S. J. E.) (Figure I, p) which is 12 mm. long, resembling a periodontal probe, rectangular in cross section and hollow ground. This instrument is used for planing the rounded corners of root surfaces.

Clinically, these instruments are used for subgingival scaling, in depth, together with a constant stream of water or selected medicaments. Since ultrasonic vibrations are sufficient to dislodge deposits, it is preferable that the instruments remain dull to avoid inadvertent overplaning of root surfaces. Radiographs (Fig. 2) of posterior teeth illustrate these instruments in place interproximally at the depth of the pocket.

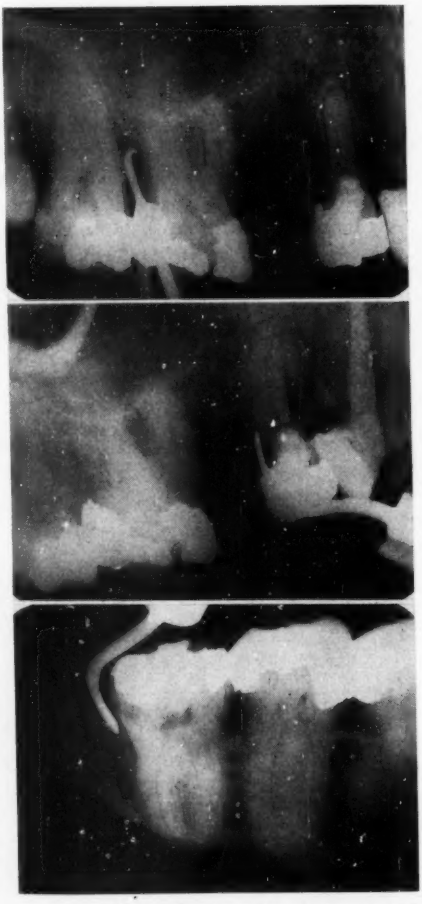

Fig. 2. Radiographs illustrating instruments at depth of pocket.

INSTRUMENTS FOR RECONTOURING RESTORATIONS

Improper contour of dental restorations of amalgam, silicate and cement still remain one of the most important environmental factors in gingivitis and periodontitis. Previous means of removing marginal overhangs, such as rotary instruments, files, chisels and abrasive strips, have had limited value. These methods have been tedious and frequently traumatic to the local tissues. A blunt chisel or sickle scaler (Fig. I, e, f, g, h) has been found to be an effective instrument to remove such excesses when used as a transducer insert with a flow of water. To illustrate this application Figure 3 shows a radiograph of a silicate on the distal of an upper right lateral incisor and an amalgam on the mesial of an upper right first bicuspid. These restorations were intentionally inserted with marginal overhangs. With a sweeping motion from tooth to restoration margin, blunt sickle scalers were vibrated and the overhangs eliminated. The removal of an overhang in an upper right first molar is shown in Figure 4.

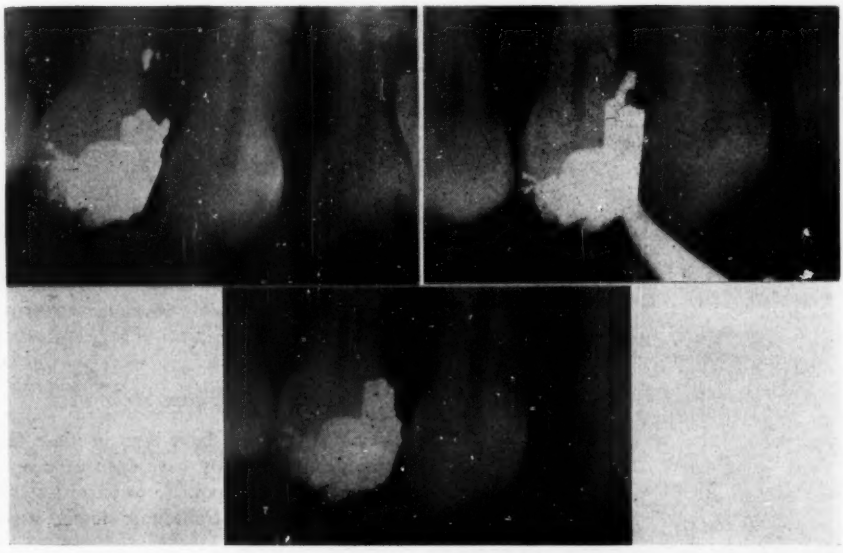

Fig. 3. Radiographs illustrating the removal of excess silicate from an incisor and excess amalgam from a bicuspid using a sickle shaped instrument.

Fig. 4. Radiographs illustrating recontouring an amalgam overhang.

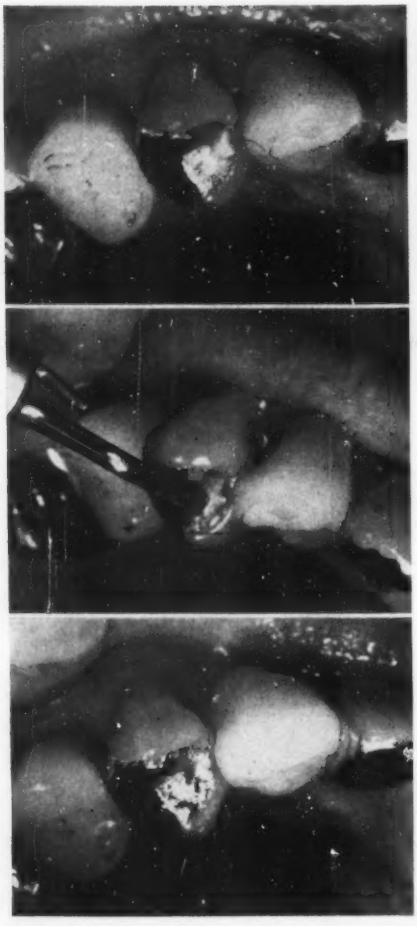

Fig. 5. Orban file burnishing gold inlay margin.

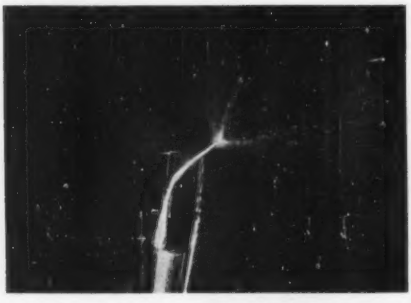

Fig. 6. Vibrating insert illustrating spray covering a wide area.

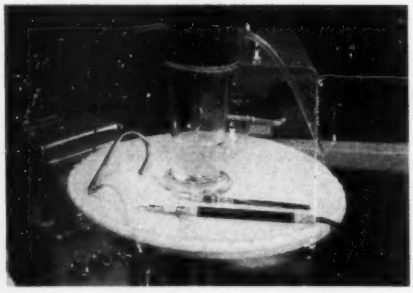

Fig. 7. Gas washing bottle set up, tubing diverting water into cuspidor and inserts.

BURNISHING GOLD MARGINS

Gold inlays that have been in place for some time frequently have open cavo-surface margins. It is possible to burnish these margins using a transducer insert with Orban files (Fig. 5).

PERIODONTAL POCKET LAVAGE

Liquids exposed to intense sonic vibrations burst into an accelerated spray covering a wide area (Fig. 6). This phenomenon of dispersion is a form of accelerated fluid jet delivery. It permits any selected solution to penetrate to the depth and extent of the periodontal pocket. In addition, bubbles are released from the fluid.[5] This combination of bubble release and spray effectively flushes debris out of the pocket.

Sodium hypochlorite solution (N. F.), available commercially as Zonite, completely dissolves necrotic tissue.[6] Its additional capacity for release of nascent gas, bubble and spray formation when exposed to the ultrasonic vibration, makes sodium hypochlorite an ideal medicament for use with this new modality.

For the purpose of medicament dispersion, ultrasonic periodontal instruments are altered. The tubing provided for the coolant water spray is diverted with a plastic tubing to drain into the cuspidor. A 28 gauge needle is used as the nozzle to direct a fine stream of fluid to the vibrating tip (Fig. I, q). The Zonite solution is used in

a dilution of one tablespoon to the quart and is supplied by means of a gas washing bottle (Eimer and Amend, Fischer-Milligan, Catalog No. 7-513) setup in a reverse direction with properly reduced fittings and tubings (Fig. 7). This bottle, used with compressed air from the dental unit, becomes a large spray bottle with fluid supply adequate for office treatment.

SUMMARY

Instruments for ultrasonic curettage, recontouring of restorations, and fluid dispersion have been described. Examples have been presented of the use of these instruments in periodontal therapy.

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The University of Washington operates on a schedule of four quarters per year, of eleven weeks each. More definite information is available in the Graduate School Bulletin. All communications should be addressed to: Saul Schluger, D.D.S., Director, Graduate Dental Education, University of Washington School of Dentistry, Seattle 5, Washington.

American Academy of Periodontology

President's Address, 1958

by DONALD A. KERR, D.D.S., *Ann Arbor, Michigan*

TO serve as the president of the American Academy of Periodontology is a great honor. I am highly appreciative of the opportunity you have afforded me to serve in this high office. I do so with pride and humility.

At this time I would like to bring to your attention the state of the Academy, to review some of the past year's activities, and to make some suggestions for the future.

In his presidential address in 1955, Dr. Harold Ray suggested a workshop to be sponsored by the Academy on the subject "Teaching of Periodontia." Dr. Ray followed up his suggestions the following year as Chairman of the Council and planned a pilot workshop as a part of the Dental Schools Meeting program. The success of this pilot study indicated the desirability of a full scale workshop on this subject. Funds for this purpose were provided by the Academy, the Proctor and Gamble Company, and the U.S.P.H.S. Dr. Ray organized the Workshop which was held in San Francisco in June of this year. The Workshop was a most successful and productive activity primarily because of the enthusiastic direction and detailed organization by Dr. Ray. The entire proceedings of the Workshop will be published and distributed to all dental schools and other interested organizations.

Because of Dr. Ray's efforts in organizing and directing this Academy-sponsored Workshop, he should be cited for meritorious service to the Academy. Because of the success of this Workshop, I recommend that the Council be empowerd to appoint a committee to plan another Academy-sponsored workshop to be held some time following the publication of the proceedings of the first workshop.

During the past year the Professional and Public Relations Committee has been very active in attempting to publicize the field of periodontology. They have also appeared before the Blatnick Committee hearing evidence on undesirable practices in dentifrice advertising. One member of the Academy, Dr. Harry Lyons, speaking for dentistry performed an outstanding service by his appearance before this Committee.

In 1955 Dr. Ray brought before the Council a motion for the organization of regional sections of the Academy. The Council did not approve his motion. Nevertheless two independent regional societies have been formed within the past year. Although these regional organizations are not sponsored by the Academy, I suggest that our members participate in the regional groups to become acquainted with their membership and thus be in a position to help guide them in their activities and be in a position to recognize talented personnel who could be recommended to the Membership Committee of the Academy.

The increase in the size of the Academy during the past few years has resulted in increased costs of operation. Rising costs of services to the Academy and the operation of the Annual Meeting have reached such proportions that it presents a considerable financial problem which must be resolved in one of two ways; either reduce the activities of the Academy and the quality of the Annual Meeting, or increase the income. I personally feel that we should be expanding our influence upon the profession and the public rather than consider curtailing our activities. If we curtail the activities, we eliminate influences on dentistry such as the effect of the recommendations of the Workshop on periodontal teaching which will have considerable impact upon the undergraduate teach-

ing of periodontics; the publication of THE JOURNAL OF PERIODONTOLOGY by the Academy which influences the practice of periodontia, spreads knowledge of the subject to the entire dental profession, and serves as a model of periodontal journalism; the activities of the Professional and Public Relations Committee which with financial assistance can do much for the Academy and dentistry.

The objectives of the Academy are: "to advance the art and science of periodontology and by its application, maintain and improve the health of the public." We are doing well in meeting these objectives by sponsoring workshops in various phases of periodontia, by being a leader in dental journalism, by expanding our professional and public relation activities, and by providing the highest caliber scientific program for our annual meetings. Therefore, I suggest to the Council and the membership that we meet the financial problem by increasing our income by an increase in dues rather than instituting an austerity program of running the Academy.

In six years the Academy will have been organized for half a century. I suggest that the Council consider the publication of a history to commemorate the 50th Anniversary of the Academy and direct the History Committee to work toward that end.

Another problem to which I would like to direct your attention is our policy in regard to acceptance of funds from commercial houses for the furtherance of Academy activities. For many years our JOURNAL has had financial difficulties which could have been solved by some advertising. It was indicated that the policy of the Academy was a desire not to accept commercial advertising so that the Academy would be free of obligation. However, when there was a need for support of the periodontal Workshop, funds were solicited from a commercial organization. At the same meeting that commercial support was announced, the Professional and Public Relations Committee asked the Council to approve a resolution to stop fraudulent advertising of dentifrices by the same house from which we had just accepted funds. Other commercial houses desire to provide funds for another workshop. Before accepting such funds, I think the Academy should establish a definite policy in this matter. Can we best meet our objectives like Jarvis in "The Devil and Daniel Webster" who sold his soul for two cents, or should we find other means of support? I think it is of extreme importance that we consider this problem carefully in all of its many manifestations and establish a definite policy which will not jeopardize the Academy in the future.

The organization of the Academy is most efficient and makes the office of president a pleasure rather than a burden. This fact is due to the effective organization of the committees. The all-important Committee on Scientific Sessions does a Herculean job in arranging the Annual Meeting. They select the Program Committee, the Local Arrangements Committee and coordinate the activities of both to provide the Academy with an outstanding Annual Meeting. I think they have done an excellent job this year, and I would like to publicly recognize them for their efforts and express my gratitude for their efforts and loyal support.

I also would like to express my sincere thanks to Stanley Baker, Chairman of the Council, for his sage advice, and to all members of the Council for their cooperation.

The assistance of our very able treasurer, Jim Aiguier, has been invaluable to me personally as well as to the Academy.

Lastly, I would like to thank our secretary and his office for making my term as president an enjoyable one. Dr. Chamberlain's attention to details, his devoted interest in the Academy, and his subtle sage advice has been of inestimable value in helping me

carry out the duties of my office. I am humbly grateful to him for all he has done for me and the Academy. You, as well as I, owe him a vote of thanks for his dedication to our organization.

With continued selection of dedicated officers and committee personnel to keep our objective always before us, the Academy will continue to grow in stature and influence.

REPORT OF THE COMMITTEE ON THE PRESIDENT'S ADDRESS

Your committee met and studied the scholarly address presented by our President Donald A. Kerr earlier this week. We were very impressed by the clear, concise and erudite manner in which Dr. Kerr set forth his thoughts relative to the activities of our Academy.

The praise extended Dr. Harold Ray for his outstanding effort in developing and directing the recent "Periodontology Workshop" in San Francisco was well deserved and we are in accord with President Kerr's statement that Dr. Ray be cited for meritorious service to the Academy.

Dr. Kerr's address included the following recommendation:

The Council be empowered to appoint a committee to plan another Academy-sponsored workshop to be held sometime following the publication of the proceedings of the first workshop.

Our Committee on the President's address asks that this recommendation be approved and that the several valuable suggestions also contained in President Kerr's address be referred to the Council for their consideration and action.

James Hollers
Delbert Nachazel
B. O. A. Thomas, Chairman

Committee Reports . . . .

NOMENCLATURE AND CLASSIFICATION COMMITTEE

Not long after the first world war, Gottlieb introduced into the periodontal literature investigative findings which indicated that periodontal diseases comprised a group of entities among which was one termed by him "diffuse atrophy." Through the subsequent years the terminology changed with the introduction of the term "periodontosis." It must be stressed, however, that the original description of the signs and symptoms of this so-called entity had undergone considerable change. Through these years much debate concerning this condition has confused rather than clarified the picture. In 1952 at the Ann Arbor, Michigan Workshop in Periodontology the question arose as to the exact nature of this manifestation and there were many who doubted its existence. Although since 1952 there has been but little written concerning this problem, one gains the distinct impression that there is no uniformity of opinion regarding the description of the clinical signs and symptoms of the condition in question. Certainly, no convincing human material of sufficient quantity has been published to establish once and for all the

pertinent histopathologic changes. It was, therefore, deemed essential by this Committee to review this subject to ascertain an analysis of current opinion and also whether any further investigative work which could shed light on this problem had been done.

In order to carry out this review, a letter and questionnaire were sent to fifty investigators. Twenty replies were received. The data sent was as follows:

"Enclosed is a summary of the statements made concerning Periodontosis at the University of Michigan Workshop in Periodontology in 1952.

"Since that time there has been meager literature concerning the consideration as to whether Periodontosis should be considered a clinical entity nor any information concerning tissue changes involved. It is believed that a review of this subject at this time is necessary.

"The Committee on Nomenclature and Classification of the Academy of Periodontology would appreciate a comment from you concerning your views. Any investigative work which you may or may not have published since 1952 should be cited. Your cooperation is appreciated."

A disease entity termed "Periodontosis" is recognized as a distinct clinical manifestation by some investigators but not by others. The entity as a whole has been differentiated from "Periodontosis." At the University of Michigan Workshop in 1952 the following statements were made by three investigators:

"Dr. Henry M. Goldman stated: Periodontosis, a disease of the attachment apparatus of the teeth, seen for the most part in very young individuals, is characterized by early loosening and migration of the teeth. The clinical behavior of this disease is such that, radiographically, bone loss takes place in a rather rapid fashion and loosening of the teeth is progressive in nature. In the initial phase, gingival changes are either absent or are of such a nature that one cannot attribute the loosening and migration of the teeth to them. The signs and symptoms of this disease, as characterized by its clinical behavior, age pattern, and radiographic changes, give rise to the opinion that such a disease process can be regarded as a clinical entity.

"Dr. Balint Orban concluded: Periodontosis is an entity, taking either the clinical or the histopathologic appearance into consideration. It is true, when the patient seeks treatment, that inflammation of the periodontal tissues is a dominating feature. However, the early development of the condition is of a degenerative nature and inflammation is a secondary occurrence.

"Inflammation is a tissue reaction of generalized nature and will present itself as a defense reaction caused by tissue damage. This tissue damage can be degeneration, which then has to be considered the primary factor and inflammation the secondary factor.

"Dr. Sigurd Ramfjord, on the basis of his research, argued: Evidence of inflammation is always present in a periodontal pocket. The retrogressive tissue changes which have been described in the few reported histologic investigations of periodontosis may be due to individual variation in inflammatory response to various types of injury. Clinical observation that periodontosis in many cases can be arrested by adequate local therapy tends to support the importance of local irritation in this disease. From the present knowledge it is not unequivocally established that periodontosis is a disease entity of pure degenerative nature, discernedly different from periodontitis."

Of the twenty replies, fourteen indicated that existence of this so-called entity; six stated that there was no foundation for the classification of the condition designated as

periodontosis. Two letters can be quoted to sum up the sentiments expressed:

"The more I see of this syndrome clinically, and I believe it is correct to call it a syndrome, the more I feel that this is an entity in periodontal disease that will reveal much more to us of the initiation of the problem of detachment than will the major complication of Periodontitis with its multitudiness contributing factors and modifications. In this direction I have accumulated a good many pure cases of the syndrome and have been gathering clinical information concerning them. I am very careful in the selection of these cases and have eliminated any that might be considered borderline and have only accepted those that fall within a definite clinical pattern.

"Very recently I was fortunate enough to find in our clinic a young man age 22, who had classic findings in regard to Periodontosis. Upon further investigation with him I found he had a twin brother, who happens to be a freshman in our dental school. Needless to say I corraled this young man immediately and found the same symptoms characteristic of his twin brother, in him. The difference only was degree of severity. Upon further investigation I found these twin brothers had two sisters and one older brother and consequently we were able to corral them and bring them in for clinical examination. They too exemplified the symptoms that we know as Periodontosis. The age range of these people is 22 to 27. Again the differences from one to another are those primarily of severity and inroads of Periodontitis superimposed upon the original entity of Periodontosis. As you can well imagine this is a jewel family find and I have even been able to corral x-rays of the parents who are both now edentulous but the x-rays that I was able to secure showed evidence also of this kind of pathology."

The second letter, in substance, stated:

"I do not believe this is a disease entity caused by systemic factors, but that these changes are produced by occlusal trauma. The changes of so-called periodontosis can be observed in those teeth which are in traumatic occlusion. In traumatism there is denegeration of periodontal membrane and also proliferation of epithelial rests. The degeneration may be fibrous, hyalin or necrotic in type. When other local factors are superimposed upon areas of degeneration, pocket formation may be rapid. These changes in association with traumatism were demonstrated in monkeys by Ramfjord.

"During the past several years we have studied continuous sections from a large number of jaws and have been more and more impressed with the degenerative changes in the periodontal membrane which are associated with other evidence of traumatism, and have been completely convinced that the changes described as periodontosis are due to traumatism. When our material is completed, we plan to publish our findings.

"I think another evidence to support this is seen in the condition designated as juvenile periodontitis or periodontosis. This condition presents a clinical picture much like the condition called periodontosis. It involves first permanent molars and incisors at an early age. One explanation for this is the fact that these are at one period in the dentition the only teeth retaining the vertical dimension. During this time they are subjected to considerable trauma, and with the superimposition of local factors periodontitis is initiated as the chief factor."

The analysis of this review revealed much pertinent data:

1. A clinical entity, the course and behavior of which seemingly falls into a definite pattern, is considered by the majority to be a factor to be considered in a classification of periodontal diseases.

2. Cases have been recorded affecting more than one offspring of a family as well as the parents. The question has been raised as to hereditary studies of such a manifestation being undertaken.

3. The observation was made that systemic factors alone do not initiate or cause periodontal pockets; this may be illustrated by many experiments. However, it is questionable that all possibilities have been explored. For example, evidences as related to protein deficiency may be an exception to the above rule.

4. Systemic factors may aggravate and accelerate tissue destruction initiated by surface irritation and occlusal trauma. Most investigators believe that this statement is true. However, still other information is necessary to corroborate this fact. Studies in wound healing are definitely necessary to establish this observation.

5. Degenerative changes in the periodontium are commonly associated with occlusal traumatism and disuse atrophy. It is also true, however, in the case of protein deprivation and ascorbic deficiency and scleroderma.

6. It has been suggested that the so-called entity periodontosis may be explained by the fact that the teeth usually affected are at one period in the formation of the dentition the only ones retaining the vertical dimension. During this time they are subjected to considerable trauma and with the added irritation of local factors, periodontitis is initiated as the chief factor thus giving rise to the syndrome usually described. It must be suggested, however, that all children go through this stage of development but do not show this pattern. The question of implicating resistance and repair as the differential must be carefully evaluated in light of the fact that examination of individuals affected by periodontosis do not exhibit any abnormal factors in the above processes.

7. It has been shown that occlusal traumatism is usually associated with a habit pattern. A good many children have habit patterns but statistically only a few cases of periodontosis have been found. These factors seemingly do not coincide.

8. All investigators agree that adequate local therapy is the only way to stop the disease process in this clinical entity. This seemingly does not coincide with a systemic causation for the disease process. However, not enough cases have been recorded or published as to long range evaluation of therapy.

Evaluation of the above statements reveals that there are many problems which still need investigation to understand the true nature of this condition. No uniformity of opinion exists concerning the course of the disease nor its reaction to therapy. Far too few cases have ever been reported which have been followed for at least a period of ten years. There is no information as to what the true clinical course has been nor whether the destructive processes continued or abated and the length of time involved. Certainly this type of investigative work is neccessary.

At present, this review certainly indicates that the classification of periodontosis as a clinical manifestation should be retained. It must be stressed, however, that it is a rare manifestation and must not be confused with the degenerative changes which comprise the clinical entity occlusal traumatism, so common a periodontal manifestation.

Harry Lyons
Joseph Bernier
Henry M. Goldman, Chairman

REPORT OF THE AMERICAN BOARD OF PERIODONTOLOGY

The American Board of Periodontology met at Indianapolis in April 1958 and examined and certified fifteen candidates. The total number of Diplomates certified is now 253. There are thirty-four applications on file of which thirty-one intend to be examined next spring so far as the Secretary knows. Indiana University will again be the host. The examination procedure consists of five case reports in by January 1, and in April a six-hour written examination, half objective type questions and half essay type, a two day clinical examination, and a half hour oral examination and interview.

Two academic years of an approved full-time post-graduate education and training program plus three years of practice devoted primarily and principally to periodontics are required as qualifications for the examination. Seventeen dental schools and eight hospital residencies in the Armed Forces and Veterans Administration meet the standards of the Council on Dental Education for post-graduate or graduate programs.

The Board has petitioned the Council on Dental Education to convene the Boards of Periodontology, Oral Pathology, Oral Medicine and Endodontics in a meeting to work out a method of combining.

The Bylaws of the Board have been amended to provide, if needed, for the appointment and service of an Executive Secretary-Treasurer, who may not be a director of the Board. The present Secretary-Treasurer is retiring at this time.

Harold J. Leonard, Secretary-Treasurer

AMERICAN ACADEMY OF PERIODONTOLOGY
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AMERICAN BOARD OF PERIODONTOLOGY

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JOURNAL OF PERIODONTOLOGY

Maynard K. Hine	Editor	(Reappointed for three year term)	1961

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	Term Expires
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(Arrangements for Annual Meeting)

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C. Mark Gilson, Chairman for '59
Zar Reader, Chairman for '60
Joe Barkley
Edgar A. Honey, Jr.

Dr. Howard A. Hartman, Cleveland
"Official Photographer"
Academy of Periodontology

Snapshots by Dr. Hartman taken during Academy meeting in Dallas, Texas

(1) Dr. Donald A. Kerr, (2) Dr. Joseph Bernier, (3) Dr. Stanley Baker, (4) Dr. Balint Orban being given the academy fellowship award by (5) Dr. Harold Ray, (6) Dr. Hunter Allen, (7) Dr. Richard Chace, (8) Dr. Sigurd Ramfjord, (9) Dr. Joseph Bernier and (10) Dr. Maynard K. Hine being photographed for television presentation in Dallas, (11) Dr. Bert Lytle, (12) Dr. Carlos Schott, (13) Dr. S. A. Schmid, (14) Dr. Sam Parks, recipient of academy fellowship award. (15) Dr. Sidney S. Friedman, Sr., (16) Dr. Sidney S. Friedman, Jr., (17) Dr. Balint Orban, guest at a testimonial dinner given November 1958 in his honor.

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Members of the Council of the American
Academy of Periodontology—1959

Front row, left to right: Drs. J. Hollers, President-elect; J. E. Aiguier, Treasurer; J. Bernier, President; D. Kerr, retiring President; S. Baker, past President; C. Chamberlain, Secretary. Second row: M. K. Hine, Editor; Williams; Miss Lorrie Hill, Secretary; C. Hopper, R. Chace, H. Archer, F. Beube, B. O. A. Thomas.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Program for the Spring Seminar

Monday, April 27

8:30 - 9:30	Registration
9:30 - 12:00	Pharmacology of Newer Drugs: Antibiotics, Muscle relaxants, Tranquilizers, etc. Henry H. Swain, M.D., University of Michigan, Ann Arbor
2:00 - 4:30	The Physiological Changes Associated with Hormonal Activity During Menstruation and Pregnancy. T. N. Evans, M.D., University of Michigan, Ann Arbor

Tuesday, April 28

9:00 - 12:00	Recent Information Relative to the Stress and Adaptation Syndrome. Robert D. Johnson, M.D., University of Michigan, Ann Arbor
2:00 - 5:00	Endocrinopathies in Relation to Oral Disease. Robert D. Johnson, M.D., University of Michigan, Ann Arbor

Wednesday, April 29

9:00 - 12:00	Periodontal Surgical Techniques. Saul Schluger, D.D.S., University of Washington, School of Dentistry, Seattle
2:00 - 3:30	Periodontal Surgical Techniques (cont.) Saul Schluger, D.D.S., University of Washington, School of Dentistry, Seattle

Only members may make application. Guests may be sponsored by a member and each member may sponsor only one guest.

For further information, write to Dr. C. M. Gilson, School of Dentistry, University of Michigan, Ann Arbor, Michigan.

Announcements . . . .

MODERN CONCEPTS REGARDING THE ETIOLOGY, DIAGNOSIS AND TREATMENT OF ORAL DISEASE

Colonel Joseph L. Bernier, Chief, Branch of Dental and Oral Pathology, Armed Forces Institute of Pathology; Pathologist, Registry of Oral Pathology, Washington, D. C., will present a course on the etiology, diagnosis and treatment of oral disease in Boston September 21, 22, 23, 1959.

In this course, Colonel Bernier will interpret diseases of the oral mucous membrane and osseus structures in terms of underlying microscopic tissue changes. The cellular and clinical changes produced by local and systemic influences will be explained. The criteria for the early diagnosis of malignancy in the oral cavity and adjacent structures will be described. In essence, this is a course designed to provide a background in the modern developments in the field of oral pathology which is indispensable for the intelligent management of oral disease. Tuition $75.00—Class Limited.

For further information and application write to Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston 11, Massachusetts.

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Professor Sidney Sorrin and associates:

Refresher course in Periodontia and Oral Medicine. Ten sessions, Wednesday, 4:00 to 6:00 p.m., February 11, 1959 to April 22, 1959. Tuition and books, $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

Periodontia and Oral Medicine in the Spanish Language. Four weeks, Monday through Friday, 9:00 a.m. to 4:00 p.m. February 23, 1959 to March 20, 1959. Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

For further information write to the Secretary, Postgraduate Studies, New York University College of Dentistry, 421 First Ave., New York, N. Y.

UNIVERSITY OF SOUTHERN CALIFORNIA

The University of Southern California School of Dentistry announces the following postgraduate courses:

. Instrumentation—Dental Hygiene. February 13-15, 1959. Fee $45—enrollment limited.

Instructors: Arnold A. Ariaudo, B.S., D.D.S., and Associates.

Through the medium of lectures and demonstrations, the following will be covered: (1) Instrumentation, (2) Oral Physiotherapy, (3) Calculus formation in relation to periodontal disease and histopathology of periodontal disease.

Laboratory phases of the course and treatment of patients will be provided.

Occlusion—Functional Development in Wax. May 8-10, 1959. Fee $80—enrollment limited.

Instructor: Everitt V. Payne, D.D.S. This course of instruction will enable the participant who becomes proficient in this technique to develop a full mouth restoration and an extremely short period of time employing the most advanced principles of generating occlusal surfaces. Harmony of form and function will be stressed. Each member of the class will work out a full mouth problem from the standpoint of articulation.

Periodontics—June 19-20, 1959. Fee $40—enrollment limited.

Instructor: Saul Schluger, D.D.S., Visiting Professor.

This two day presentation will be oriented toward establishing a rational approach to a rather broad spectrum of periodontal techniques. In addition to the basic methods of subgingival curettage, the following will be presented with emphasis on indication as well as method: Gingivectomy, Occlusal adjustment, more recent advances in periodontal surgery, Frenectomy, Osseous resection, Infrabony techniques, and Mucogingival surgery.

For further information write University of Southern California School of Dentistry Postgraduate Division, 925 West 34th Street, Los Angeles 7, California.

THE UNIVERSITY OF KANSAS CITY

The University of Kansas City will present the following courses:

Oral diagnosis and oral medicine, by Dr. H. B. G. Robinson. This course is designed for the general practitioner in the field of oral diagnosis and in relationship of systemic and oral diseases. Comprehensive oral diagnosis, treatment planning, and diagnostic methods will be emphasized. Fee $25. March 5.

Periodontics, by Dr. Frank Buebe, New York City, New York. This course is designed to assist the general practitioner in making a diagnosis, planning treatment and the use of adjunctive drugs in the restoration of the patient to good gingival health. Fee $50. April 1-2.

For further information, write to Postgraduate Division, School of Dentistry, The University of Kansas City, 1108 East Tenth Street, Kansas City 6, Missouri.

UNIVERSITY OF ALABAMA

The University of Alabama School of Dentistry will present three refresher courses during the month of February. Miss Gertrude Sinnett, B.S., D.H., will conduct a course in "Dental Assisting" on February 14 and 15; Irving R. Hardy, D.M.D., will present a program in "A Practical Approach to the Full Denture Problem" on February 21, 22, and 23; and Samuel D. Gore, D.D.S., will instruct a program in "Removable Orthodontic Appliance Construction" on February 28, March 1 and 2. (This course is limited to practicing orthodontists.)

A program in "Occlusal Adjustment: Its relation to dental, periodontal and temporomandibular joint problems" will be presented by Sigurd Ramfjord, L.D.S., M.S., Ph.D., on March 7, 8, and 9, 1959.

Two courses are planned for the month of April. On April 3, 4, and 5, "Partial Denture Prosthesis" will be conducted by William L. McCracken, D.D.S., M.S.; and on April 10, 11, and 12, "Oral Medicine" will be offered by Emanuel Cheraskin, D.M.D., M.D.

For further information, please contact the Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Avenue South, Birmingham, Alabama.

TUFTS UNIVERSITY

Tufts University School of Dental Medicine announces the following postgraduate courses:

DPG 606—Clinical Adjustment of the Occlusion, April 2, 3, 4, 1959. Tuition $100—class limited. Dr. Bernard Jankelson, Diplomate American Board of Prosthodontics, Clinical Associate and Lecturer University of Washington.

The first day will be devoted to clarification of the physiology of the stomatognathic area, particularly as it pertains to the occlusion of the teeth. It will be shown that as the result of this information our concepts of the basic nature of the dentition and of occlusion must be considerably modified. The remainder of the course will demonstrate the clinical adjustment of a series of cases by means of television projection. The clinical diagnosis and treatment will be done directly in the mouth. A sufficient number of cases will be done to cover the various conditions encountered in practice.

DPG 601—A special participation course in Periodontology. September 21-25, 1959. Tuition $150—class limited. (Upon inquiry special tuition can be arranged for dentists from outside U. S.).

In this course emphasis is placed upon learning by clinical experience, in addition to lectures and demonstrations. Half the time is devoted to clinical sessions in which each participant treats patients under Dr. Glickman's guidance. Bifurcation involvement, infrabony pockets, the "unembellished gingivectomy" frenectomy and splinting are dealt with at the chairside. The "Bone Factor" concept for the diagnosis, determination of prognosis, and treatment planning for individual teeth and the dentition as a functioning unit will be explained in detail.

For further information and application write to Division of Graduate and Postgraduate Studies, Tufts University School of Dental Medicine, 136 Harrison Avenue, Boston, Mass.

BOSTON UNIVERSITY

The Department of Stomatology, Division of Graduate Studies, School of Medicine, Boston University announces the following postgraduate courses:

Advanced Course in Periodontal Therapy, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin and Staff. March 2-7, 1959.

Periodontal Therapy, Dr. Henry M. Goldman, Dr. Bernard S. Chaikin and Staff. March 16-27, 1959.

Occlusal Adjustment, Dr. Bernard S. Chaikin, Dr. Henry M. Goldman and Staff, April 27-May 1, 1959.

Periodontal Prosthesis, Dr. Lee Talkov, Dr. David J. Baraban, May 18-22, 1959.

Surgical Technics Employed in Periodontal Therapy, Dr. Lewis Fox, May 25-29, 1959.

Periodontal Pathology, Dr. Henry Goldman, Dr. Jack Bloom and Staff, June 8 to 12, 1959.

For further information write to Director of Postgraduate Studies, Department of Stomatology, Massachusetts Memorial Hospital, 750 Harrison Avenue, Boston 18, Massachusetts.

NORTHWESTERN UNIVERSITY

Northwestern University Dental School announces the following postgraduate course:

February 2-6, 1959: Newer Concepts in Periodontics. Tuition $150.

Faculty: Max Gratzinger, M.D., D.D.S., M.S., Associate Professor Emeritus of Oral Pathology (Course Leader), Stanley C. Harris, Ph.D., Professor of Physiology and Pharmacology, Irvin T. Harris, D.D.S., M.S.D., Associate Professor of Oral Pathology, Marjorie Houston, D.M.D., M.S., Associate Professor of Oral Pathology and Harold T. Perry, D.D.S., M.S.D., Instructor in Orthodontics.

This one week course will undertake to acquaint the participants with an approach to the theory and practice of periodontics which deviates in important details from other presentations.

For further information write to Director of Dental Postgraduate Study, Northwestern University Dental School, 311 East Chicago Avenue, Chicago 11, Illinois.

Letters to the Editor . . . .

I wish to comment on a letter to the Editor by Dr. John Oppie McCall which appeared in the October issue of the Journal of Periodontology concerning my paper "Reattachment and Roentgenograms."

I think that Dr. McCall misunderstood the purpose of the paper and some of the opinions expressed in it. First, may I point out that it was not my intent to impugn anyone's ability or to disparage any man's work. It was merely an effort to examine my own thinking and work, as well as that of others, in order to evaluate the results honestly but critically. In addition it attempted to offer an explanation for the seemingly para-

doxical results I found roentgenographically after performing gingival and osseous surgery.

Dr. McCall wrote "I think it is highly unfortunate for a Periodontist of Dr. Friedman's ability and standing to take the negative attitude expressed in his article." My attitude was not negative. On the contrary, it was positive but with reservations born of curiosity about the realistic aspect of success in reattachment therapy.

Following are some quotes from my paper: "That reattachment can be attained is *indisputable,* but the *amount* is open to question. Recent evidence points to greater and more frequent success." I cited three cases of my own that I believe to be successful cases of reattachment as shown in Figures 6, 8 and 9. On p. 108 I wrote, "Both human and animal experimental data prove that reattachment is possible. However, as yet this data provides no statistically valid clue as to the frequency of its occurrence and the amount of reattachment. The histologic evidence offered shows the average amount of reattachment to be 2-3 mm. The clinico-roentgenographic evidence indicates greater success." On p. 105, "The purpose of this paper is to focus attention on the need for sharp, critical evaluation of the results obtained when reattachment is attempted." On p. 101, "One must exercise caution and be keenly perceptive to interpret accurately from the roentgenogram what bone changes have occurred. The best method available at this time to arrive at a presumptive conclusion that connective tissue reattachment has been achieved is by the correlation between clinical measurements and roentgenographic changes. A conclusion reached in this way *may be valid if the clinician is knowing and precise.*"

Dr. McCall wrote, "He states that while remaining bone becomes more dense after treatment it does not advance coronally beyond its previous level." On p. 101 what I wrote was, "The apparent regeneration of the alveolar crest in a coronal direction in the roentgenogram does not *always* signify that this has occurred." Dr. McCall wrote. "With regard to the probe . . . but the periodontist who sees his patients routinely over a period of many years must face recurrences of disease if he has failed to secure real attachment." I agree with Dr. McCall. On p. 108 I wrote, "However, if such measurements (probing) seem to indicate new attachment and a critical assay of the roentgenogram parallels the clinical finding, we may assume that reattachment has occurred. Probing such areas repeatedly over a period of years may provide corroborative evidence that reattachment has been gained."

I think Dr. McCall misunderstood an important phase of my paper. He infers in his letter that I performed gingivectomy and osseous surgery to gain reattachment. I expected no reattachment from these operations and did not get any. *This was precisely the point of my paper.* It was in those cases where no reattachment was attempted or achieved that the roentgenographic changes appeared so startling. It appeared as if bone had regenerated in a coronal direction. These changes were more dramatic than many roentgenograms offered as proof of reattachment. It was because of these changes and my need to find an answer for them that my paper was written.

I have learned much from my attempt to interpret the puzzling roentgenographic changes following my cases. The writing of "Reattachment and Roentgenograms" was motivated by this knowledge and the hope that it would help others. It was not motivated to disparage the subject of reattachment or the results of the clinicians and experimenters who have reported on this subject.

Nathan Friedman, D.D.S.
436 North Roxbury Dr.
Beverly Hills, Calif.

To the Editor:

I have read with great interest the article "Periodontosis in a Child with Hyperkeratosis Palmo-Plantaris" by Dekker and Jansen in the October issue.

There is a remarkable similarity between the condition described in this article and that of several children described by Warkany and others. These authors have described a condition occurring in children ranging in age from 11 months to 13 years with hyperkeratosis and desquamation of their palms and soles of their feet similar to the hyperkeratosis palmo-plantaris described by Dekker and Jansen. Periodontosis and/or shedding of teeth in such patients is only infrequently described in the periodical literature although the textbook descriptions list them as common findings. The condition which Warkany and others have described is known as acrodynia or pink disease and is felt by many to be the result of a hypersensitivity to mercury or other heavy metal. Acrodynia is not generally considered to have a familial basis and thus is in contrast to the condition reported by Dekker and Jansen which apparently has a significant familial pattern.

It would be most interesting in view of the periodontal significance of this condition to solicit the experience of other dentists and periodontists who may have seen similar problems in children in their practices.

I will appreciate hearing from all of your readers who have had an opportunity to examine patients with conditions of this sort.

Roland R. Hawes, D.M.D.
Supervisor of Clinics, Eastman Dental Dispensary, 800 Main Street East, Rochester, N. Y.

Letter from the Editor:

It has been said that an institution is the lengthened shadow of one man. Perhaps it can also be said that a profession is but the lengthened shadows of many men; the dental profession has been particularly fortunate in having outstanding leaders who over the years have been dedicated to the elevation of dental science. The "lengthened shadows" of these leaders have been consolidated by the shadows of many unheralded dentists who have devoted their entire time, energy and ability to the conscientious practice of dentistry. Such an individual was my father, Dr. Clyde L. Hine, general practitioner of dentistry in Tuscola, Illinois, who died January 3, 1959 after a short illness. Dentistry owes much to dedicated general practitioners such as my father. We wish to thank our many friends who were kind enough to send flowers and express sympathy.

To all our readers we express our regrets that this Journal is late, but we think our alibi is sound.

Maynard K. Hine, D.D.S.
Editor